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☐	L19	L14 and specific	35
☐	L18	L17 and VSV	0
☐	L17	Purging and l13	11
☐	L16	l13 and oncolytic	3
☐	L15	L14 and cancer	29
☐	L14	L13 and VSV	35
☐	L13	L12 and l1	277
☐	L12	ex adj vivo	34639
☐	L11	L2 and specific adj killing	1
☐	L10	L9 and VSV	1
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☐	L8	L2 and autologous	15
☐	L7	L5 and leukemia	2
☐	L6	L4 and autologous	13
☐	L5	L4 and hematopoeitic	2
☐	L4	L2 and 435/320.1.ICLS.	46
☐	L3	L2 and oncolytic	1
☐	L2	L1 and VSV	56
☐	L1	424/93.1.ICLS.	992

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Search	Most Recent Queries	Time	Result
#18	Search **interferon inresponsiveness of tumor cell and VSV treatment**	13:58:50	56
#16	Search **interferon inresponsiveness of tumor cell and VSV**	13:42:01	107
#15	Search **interferon inresponsiveness of tumor cell**	13:41:44	29533
#10	Search **interferon inresponsiveness of tumor and oncolytic**	13:37:48	47
#9	Search **interferon inresponsiveness of tumor and leukemia**	13:36:27	5654
#8	Search **interferon inresponsiveness of tumor and leukimia**	13:36:19	38565
#7	Search **interferon inresponsiveness of tumor**	13:35:59	38565
#4	Search **viral CD30 and inflammation**	08:27:15	7
#3	Search **CD30 and inflammation**	08:27:06	60
#1	Search **TNF type II receptor and CD30**	08:22:11	19

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Search	Most Recent Queries	Time	Result
#27	Search **interferon unresponsive and VSV** Limits: **Entrez Date to 2000/06/26**	14:08:35	0
#26	Search **interferon unresponsive and VSv** Limits: **Entrez Date to 2000/06/26**	14:08:30	0
#25	Search **interferon unresponsive and leukemia** Limits: **Entrez Date to 2000/06/26**	14:07:30	27
#24	Search **interferon unresponsive and leukemia**	14:07:08	33
#23	Search **interferon inresponsive and leukemia**	14:07:06	0
#18	Search **interferon inresponsiveness of tumor cell and VSV treatment**	13:58:50	56
#16	Search **interferon inresponsiveness of tumor cell and VSV**	13:42:01	107
#15	Search **interferon inresponsiveness of tumor cell**	13:41:44	29533
#10	Search **interferon inresponsiveness of tumor and oncolytic**	13:37:48	47
#9	Search **interferon inresponsiveness of tumor and leukemia**	13:36:27	5654
#8	Search **interferon inresponsiveness of tumor and leukimia**	13:36:19	38565
#7	Search **interferon inresponsiveness of tumor**	13:35:59	38565
#4	Search **viral CD30 and inflammation**	08:27:15	7
#3	Search **CD30 and inflammation**	08:27:06	60
#1	Search **TNF type II receptor and CD30**	08:22:11	19

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=> VSV
L1 5131 VSV

=> oncolytic
L2 2240 ONCOLYTIC

=> L1 and L2
L3 58 L1 AND L2

=> hematopoeitic
L4 189 HEMATOPOEITIC

=> L4 and l3
L5 0 L4 AND L3

=> vivo
L6 873965 VIVO

=> L6 and L3
L7 25 L6 AND L3

=> D L7 IBIB ABS 1-25

L7 ANSWER 1 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2006:52327 CAPLUS
DOCUMENT NUMBER: 144:425241
TITLE: Oncolytic activity of vesicular stomatitis virus in primary adult T-cell leukemia
AUTHOR(S): Cesaire, R.; Oliere, S.; Sharif-Askari, E.; Loignon, M.; Lezin, A.; Olindo, S.; Panelatti, G.; Kazanji, M.; Aloyz, R.; Panasci, L.; Bell, J. C.; Hiscott, J.
CORPORATE SOURCE: Laboratoire de Virologie-Immunologie and, Centre Hospitalier Universitaire de Fort-de-France, Martinique, Fr.
SOURCE: Oncogene (2006), 25(3), 349-358
CODEN: ONCNES; ISSN: 0950-9232
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Treatments for hematol. malignancies have improved considerably over the past decade, but the growing therapeutic arsenal has not benefited adult T-cell leukemia (ATL) patients. Oncolytic viruses such as vesicular stomatitis virus (VSV) have recently emerged as a potential treatment of solid tumors and leukemias in vitro and in vivo. In the current study, we investigated the ability of VSV to lyse primary human T-lymphotropic virus type 1 (HTLV-1)-infected T-lymphocytes from patients with ATL. Ex vivo primary ATL cells were permissive for VSV and underwent rapid oncolysis in a time-dependent manner. Importantly, VSV infection showed neither viral replication nor oncolysis in HTLV-1-infected, nonleukemic cells from patients with HTLV-1-assocd. myelopathy/tropical spastic paraparesis (HAM/TSP), and in naive CD4+ T-lymphocytes from normal individuals or in ex vivo cell samples from patients with chronic lymphocytic leukemia (CLL). Interestingly, activation of primary CD4+ T-lymphocytes with anti-CD3/CD28 monoclonal antibody, and specifically with anti-CD3, was sufficient to induce limited viral replication and oncolysis. However, at a similar level of T-cell activation, VSV replication was increased fourfold in ATL cells compared to activated CD4+ T-lymphocytes, emphasizing the concept that VSV targets genetic defects unique to tumor cells to facilitate its replication. In conclusion, our findings provide the first essential information for the development of a VSV-based treatment for ATL.
REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 2 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:421155 CAPLUS
DOCUMENT NUMBER: 143:93884
TITLE: Targeting human glioblastoma cells: Comparison of nine viruses with oncolytic potential
AUTHOR(S): Wollmann, Guido; Tattersall, Peter; van den Pol, Anthony N.
CORPORATE SOURCE: Department of Neurosurgery, Yale University School of Medicine, New Haven, CT, 06520, USA
SOURCE: Journal of Virology (2005), 79(10), 6005-6022
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Brain tumors classified as glioblastomas have proven refractory to treatment and generally result in death within a year of diagnosis. The authors used seven in vitro tests and one in vivo trial to compare the efficacy of nine different viruses for targeting human glioblastoma. Green fluorescent protein (GFP)-expressing vesicular stomatitis (VSV), Sindbis virus, pseudorabies virus (PRV), adeno-assocd. virus (AAV), and minute virus of mice i-strain (MVMi) and MVMp all infected glioblastoma cells. Mouse and human cytomegalovirus, and simian virus 40 showed only low levels of infection or GFP expression. VSV and Sindbis virus showed strong cytolytic actions and high rates of replication and spread, leading to an elimination of glioblastoma. PRV and both MVM strains generated more modest lytic effects and replication capacity. VSV showed a similar oncolytic profile on U-87 MG and M059J glioblastoma. In contrast, Sindbis virus showed strong preference for U-87 MG, whereas MVMi and MVMp preferred M059J. Sindbis virus and both MVM strains showed highly tumor-selective actions in glioblastoma plus fibroblast coculture. VSV and Sindbis virus were serially passaged on glioblastoma cells; we isolated a variant, VSV-rp30, that had increased selectivity and lytic capacity in glioblastoma cells. VSV and Sindbis virus were very effective at replicating, spreading within, and selectively killing human glioblastoma in an in vivo mouse model, whereas PRV and AAV remained at the injection site with minimal spread. Together, these data suggest that four (VSV, Sindbis virus, MVMi, and MVMp) of the nine viruses studied merit further anal. for potential therapeutic actions on glioblastoma.
REFERENCE COUNT: 67 THERE ARE 67 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 3 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:953474 CAPLUS
TITLE: Sensitivity of prostate tumors to wild type and M protein mutant vesicular stomatitis viruses
AUTHOR(S): Ahmed, Maryam; Cramer, Scott D.; Lyles, Douglas S.
CORPORATE SOURCE: Department of Biochemistry, Wake Forest University School of Medicine, Winston-Salem, NC, 27157, USA
SOURCE: Virology (2004), 330(1), 34-49
CODEN: VIRLAX; ISSN: 0042-6822
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Because of its potent ability to induce apoptosis, vesicular stomatitis virus (VSV) is an attractive candidate as an oncolytic virus for tumor therapy. Previous studies have suggested that VSV selectively infects tumor cells due to defects in their antiviral responses making them more susceptible to VSV infection than normal cells. We tested this hypothesis in the prostate tumor system by comparing LNCaP and PC-3 prostate tumor cells to benign human prostatic epithelial cells from patient prostatectomy specimens. We compared the cell killing ability of a recombinant virus contg. a wild-type (wt) M protein (rwt) and an isogenic M protein mutant virus (rM51R-M) that induces interferon (IFN) in infected cells and should display a greater selectivity for tumor cells. Our results showed that in single-cycle infection expts., LNCaP cells were sensitive to killing by both wt and mutant viruses, while PC-3 cells were highly resistant to VSV -induced cell killing. LNCaP and benign prostate cells were similarly susceptible to both viruses, indicating that normal prostate cells are not inherently resistant to killing by VSV. In each of the cell lines, the rM51R-M virus induced similar levels of apoptosis to rwt virus, showing that the M protein does not play a significant role in apoptosis induction by VSV in these cells. In multiple-cycle infection expts., LNCaP cells were more sensitive than benign prostatic epithelial cells to virus-induced cell killing by rM51R-M virus, but not rwt virus. Both viruses were equally effective at reducing LNCaP tumor vol. in vivo following intratumoral and i.v. inoculation in nude mice, while PC-3 tumors were resistant to VSV treatment. None of the mice treated with rM51R-M virus died as a result of virus infection, while 50-71% of mice treated with rwt virus succumbed to virus infection. Similarly, when inoculated by the more sensitive intranasal route, the rM51R-M virus was less pathogenic than the rwt virus from which it was derived. These results indicate that M protein mutant viruses are superior candidates as oncolytic viruses for therapies of prostate tumors, but future strategies for use of VSV will require testing individual tumors for their susceptibility to virus infection.

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L7 ANSWER 4 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:824043 CAPLUS
DOCUMENT NUMBER: 141:325690
TITLE: VSV mutants contg. mutations in matrix protein capable of stimulating cytokine prodn. and shutting down innate immunity and use thereof as vaccines and anti-cancer agents
INVENTOR(S): Bell, John C.; Lichty, Brian D.; Stojdl, David F.
PATENT ASSIGNEE(S): Ottawa Health Research Institute, Can.; Wellstat Biologics Corporation
SOURCE: PCT Int. Appl., 93 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004085659	A2	20041007	WO 2004-CA463	20040329
WO 2004085659	A3	20041209		
CN 1788088	A	20060614	CN 2004-80008416	20040329

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2003-457591P P 20030327
AB The present invention provides mutant viruses with a decreased ability to block nuclear transport of mRNA or protein in an infected cell which are attenuated in vivo. The mutant viruses of the present invention may also be capable of triggering the anti-viral systems of normal host cells while remaining sensitive to the effects of these systems. The mutant viruses contain single, double or triple mutation(s) in matrix protein, such as M51R, M51A, M51-54A, .DELTA.M51, .DELTA.M51-54,

.DELTA.M51-57, V221F, S226R, .DELTA.V221-S226, M51X, V221X, and S226X. In particular embodiments, interferon .beta. stimulation and oncolytic activity were demonstrated by two specific mutants AV1 (T1026R) and AV2 (TP3) of the Indiana serotype of VSV, which are are selectively attenuated in interferon-responsive cells. AV1 and AV2 were tested in a xenograft model of human ovarian cancer and in an immune competent mouse model of metastatic colon cancer. While highly attenuated for growth in normal mice, both AV1 and AV2 effected complete and durable cures in the majority of treated animals when delivered systemically. The present invention further provides for the use of the mutant viruses in a range of applications including, but not limited to, as therapeutics for the treatment of cancer and infections, as vaccines and adjuvants, as viral vectors, and as oncolytic and cytolytic agents for the selective lysis of malignant or infected cells.

L7 ANSWER 5 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:824042 CAPLUS
DOCUMENT NUMBER: 141:325689
TITLE: Mutant vesicular stomatitis viruses containing mutations in matrix protein capable of stimulating cytokine production and shutting down innate immunity and use thereof as vaccines and anti-cancer agents
INVENTOR(S): Bell, John C.; Lichty, Brian D.; Stojdl, David F.
PATENT ASSIGNEE(S): Ottawa Health Research Institute, Can.; Wellstat Biologics Corporation
SOURCE: PCT Int. Appl., 106 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004085658	A1	20041007	WO 2004-CA460	20040329

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN,

TD, TG
AU 2004223808 A1 20041007 AU 2004-223808 20040329
CA 2520279 AA 20041007 CA 2004-2520279 20040329
EP 1606411 A1 20051221 EP 2004-723949 20040329
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK
CN 1788088 A 20060614 CN 2004-80008416 20040329
PRIORITY APPLN. INFO.: US 2003-457591P P 20030327
WO 2004-CA460 W 20040329

AB The present invention provides mutant viruses with a decreased ability to block nuclear transport of mRNA or protein in an infected cell which are attenuated in vivo. The mutant viruses of the present invention may also be capable of triggering the anti-viral systems of normal host cells while remaining sensitive to the effects of these systems. The mutant viruses contain single, double or triple mutation(s) in matrix protein, such as M51R, M51A, M51-54A, .DELTA.M51, .DELTA.M51-54, .DELTA.M51-57, V221F, S226R, .DELTA.V221-S226, M51X, V221X, and S226X. In particular embodiments, interferon .beta. stimulation and oncolytic activity were demonstrated by two specific mutants AV1 (T1026R) and AV2 (TP3) of the Indiana serotype of VSV, which are are selectively attenuated in interferon-responsive cells. AV1 and AV2 were tested in a xenograft model of human ovarian cancer and in an immune competent mouse model of metastatic colon cancer. While highly attenuated for growth in normal mice, both AV1 and AV2 effected complete and durable cures in the majority of treated animals when delivered systemically. The present invention further provides for the use of the mutant viruses in a range of applications including, but not limited to, as therapeutics for the treatment of cancer and infections, as vaccines and adjuvants, as viral vectors, and as oncolytic and cytolytic agents for the selective lysis of malignant or infected cells.

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L7 ANSWER 6 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:725783 CAPLUS
TITLE: Vesicular Stomatitis Virus: A Potential Therapeutic Virus for the Treatment of Hematologic Malignancy
AUTHOR(S): Lichty, Brian D.; Stojdl, David F.; Taylor, Rebecca A.; Miller, Leigh; Frenkel, Irina; Atkins, Harold; Bell, John C.
CORPORATE SOURCE: Ottawa Regional Cancer Centre Research Laboratories, Ottawa, ON, K1H 1C4, Can.
SOURCE: Human Gene Therapy (2004), 15(9), 821-831
CODEN: HGTHE3; ISSN: 1043-0342

PUBLISHER: Mary Ann Liebert, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Certain strains of vesicular stomatitis virus (VSV) have been shown to be oncolytic in a wide variety of solid tumors. In the present study, we tested the leukemolytic properties of VSV using established leukemia cell lines and primary patient material. VSV efficiently killed essentially all leukemic cell lines. In contrast, however, normal clonogenic bone marrow progenitor cells and peripheral blood cells were remarkably refractory to infection by VSV. By exploiting this large difference in susceptibility to infection we successfully purged contaminating leukemic cells from cultures of peripheral blood progenitor cells (PBPC) using VSV. VSV was also able to infect and kill leukemic cells in primary samples taken from patients with multiple myeloma (MM). This study demonstrates the potential utility of VSV in the treatment, both ex vivo and in vivo, of hematol. malignancies.
REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 7 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:706181 CAPLUS
DOCUMENT NUMBER: 141:346449
TITLE: Replication and cytopathic effect of oncolytic vesicular stomatitis virus in hypoxic tumor cells in vitro and in vivo
AUTHOR(S): Connor, John H.; Naczki, Christine; Koumenis, Costas; Lyles, Douglas S.
CORPORATE SOURCE: Department of Biochemistry, Wake Forest University School of Medicine, Winston-Salem, NC, USA
SOURCE: Journal of Virology (2004), 78(17), 8960-8970
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Tumor hypoxia presents an obstacle to the effectiveness of most antitumor therapies, including treatment with oncolytic viruses. In particular, an oncolytic virus must be resistant to the inhibition of DNA, RNA, and protein synthesis that occurs during hypoxic stress. Here, the authors show that vesicular stomatitis virus (VSV), an oncolytic RNA virus, is capable of replication under hypoxic conditions. In cells undergoing hypoxic stress, VSV infection produced larger amts. of mRNA than under normoxic conditions. However, translation of these mRNAs was reduced at earlier times postinfection in hypoxia-adapted cells than in normoxic cells. At later

times postinfection, VSV overcame a hypoxia-assocd. increase in .alpha. subunit of eukaryotic initiation factor 2 (eIF-2.alpha.) phosphorylation and initial suppression of viral protein synthesis in hypoxic cells to produce large amts. of viral protein. VSV infection caused the dephosphorylation of the translation initiation factor eIF-4E and inhibited host translation similarly under both normoxic and hypoxic conditions. VSV produced progeny virus to similar levels in hypoxic and normoxic cells and showed the ability to expand from an initial infection of 1% of hypoxic cells to spread through an entire population. In all cases, virus infection induced classical cytopathic effects and apoptotic cell death. When VSV was used to treat tumors established in nude mice, the authors found VSV replication in hypoxic areas of these tumors. This occurred whether the virus was administered intratumorally or i.v. These results show for the first time that VSV has an inherent capacity for infecting and killing hypoxic cancer cells. This ability could represent a crit. advantage over existing therapies in treating established tumors.

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L7 ANSWER 8 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:534872 CAPLUS

DOCUMENT NUMBER: 142:16351

TITLE: Recombinant vesicular stomatitis virus vectors as oncolytic agents in the treatment of high-grade gliomas in an organotypic brain tissue slice-glioma coculture model

AUTHOR(S): Duntsch, Christopher D.; Zhou, Qihong; Jayakar, Himangi R.; Weimar, James D.; Robertson, Jon H.; Pfeffer, Lawrence M.; Wang, Lie; Xiang, Zixiu; Whitt, Michael A.

CORPORATE SOURCE: Departments of Neurosurgery, Pathology and Laboratory Medicine, and Molecular Sciences, The University of Tennessee Health Science Center, Memphis, TN, USA

SOURCE: Journal of Neurosurgery (2004), 100(6), 1049-1059 CODEN: JONSAC; ISSN: 0022-3085

PUBLISHER: American Association of Neurological Surgeons

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Object: The purpose of this study was to evaluate both replication-competent and replication-restricted recombinant vesicular stomatitis virus (VSV) vectors as therapeutic agents for high-grade gliomas by using an organotypic brain tissue slice-glioma coculture system. Methods: The coculture system involved growing different brain structures together to allow neurons from these tissues to

develop synaptic connections similar to those found in vivo. Rat C6 or human U87 glioma cells were then introduced into the culture to evaluate VSV as an oncolytic therapy. The authors found that recombinant wild-type VSV (rVSV-wt) rapidly eliminated C6 glioma cells from the coculture, but also caused significant damage to neurons, as measured by a loss of microtubule-assocd. protein 2 immunoreactivity and a failure in electrophysiol. responses from neurons in the tissue slice. Nonetheless, pretreatment with interferon beta (IFN.beta.) virtually eliminated VSV infection in healthy tissues without impeding any oncolytic effects on tumor cells. Despite the protective effects of the IFN.beta. pretreatment, the tissue slices still showed signs of cytopathol. when exposed to rVSV-wt. In contrast, pretreatment with IFN.beta. and inoculation with a replication-restricted vector with its glycoprotein gene deleted (rVSV-.DELTA.G) effectively destroyed rat C6 and human U87 glioma cells in the coculture, without causing detectable damage to the neuronal integrity and electrophysiol. properties of the healthy tissue in the culture. Conclusions: Data in this study provide in vitro proof-of-principle that rVSV-.DELTA.G is an effective oncolytic agent that has minimal toxic side effects to neurons compared with rVSV-wt and therefore should be considered for development as an adjuvant to surgery in the treatment of glioma.

REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 9 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:363266 CAPLUS

DOCUMENT NUMBER: 140:417458

TITLE: Syncytia Induction Enhances the Oncolytic Potential of Vesicular Stomatitis Virus in Virotherapy for Cancer

AUTHOR(S): Ebert, Oliver; Shinozaki, Katsunori; Kournioti, Chryssanthi; Park, Man-Seong; Garcia-Sastre, Adolfo; Woo, Savio L. C.

CORPORATE SOURCE: Carl C. Icahn Center for Gene Therapy and Molecular Medicine, Mount Sinai School of Medicine, New York, NY, 10029, USA

SOURCE: Cancer Research (2004), 64(9), 3265-3270 CODEN: CNREA8; ISSN: 0008-5472

PUBLISHER: American Association for Cancer Research

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Vesicular stomatitis virus (VSV) selectively replicates in tumor but not in normal cells and is being developed as an oncolytic agent for cancer therapy. Here we report the construction of a

recombinant VSV capable of inducing syncytia formation between tumor cells through membrane fusion at neutral pH, which led to enhanced oncolytic properties against multifocal hepatocellular carcinoma (HCC) in the livers of immunocompetent rats. Recombinant VSV vectors were constructed by insertion into their genome a transcription unit expressing a control or fusion protein derived from Newcastle disease virus. In vitro characterization of the recombinant fusogenic VSV vector on human and rat HCC cells showed extensive syncytia formation and significantly enhanced cytotoxic effects. In vivo, administration of fusogenic VSV into the hepatic artery of Buffalo rats bearing syngeneic multifocal HCC lesions in their livers resulted in syncytia formation exclusively within the tumors, and there was no collateral damage to the neighboring hepatic parenchyma. The fusogenic VSV also conferred a significant survival advantage over a nonfusogenic control virus in the treated animals ($P = 0.0078$, log-rank test). The results suggest that fusogenic VSV can be developed into an effective and safe therapeutic agent for cancer treatment in patients, including those with multifocal HCC in the liver.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 10 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2003:982369 CAPLUS

DOCUMENT NUMBER: 140:58249

TITLE: The Oncolytic Effect of Recombinant Vesicular Stomatitis Virus Is Enhanced by Expression of the Fusion Cytosine Deaminase/Uracil Phosphoribosyltransferase Suicide Gene

AUTHOR(S): Porosnicu, Mercedes; Mian, Abdul; Barber, Glen N.

CORPORATE SOURCE: Department of Microbiology and Immunology and Sylvester Comprehensive Cancer Center, University of Miami School of Medicine, Miami, FL, USA

SOURCE: Cancer Research (2003), 63(23), 8366-8376
CODEN: CNREA8; ISSN: 0008-5472

PUBLISHER: American Association for Cancer Research

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Vesicular stomatitis virus (VSV) has recently been demonstrated to exhibit significant oncolytic capabilities against a wide variety of tumor models in vitro and in vivo. To potentially enhance the oncolytic effect, we generated a novel recombinant VSV (rVSV) that expressed the fusion suicide gene Escherichia coli cytosine deaminase (CD)/uracil phosphoribosyltransferase (UPRT). RVSV encoding the CD/UPRT fusion gene (VSV-C:U) exhibited normal growth properties and generated high levels of biol. active CD/UPRT that

could catalyze the modification of 5-fluorocytosine into chemotherapeutic 5-fluorouracil (5-FU), which exhibited considerable bystander effect. Intratumoral inoculation of VSV-C:U in the presence of the systemically administered prodrug 5-fluorocytosine produced statistically significant redns. in the malignant growth of syngeneic lymphoma (A20) or mammary carcinoma (TSA) in BALB/c mice compared with rVSV treatments or with control 5-FU alone. Aside from detecting prolonged therapeutic levels of 5-FU in VSV-C:U-treated animals harboring TSA tumors and enhancing bystander killing of tumor cells, we demonstrated marked activation of IFN-.gamma.-secreting cytotoxic T cells by enzyme-linked immunospot anal. that may have also facilitated tumor killing. In conclusion, the insertion of the fusion CD/UPRT suicide gene potentiates the oncolytic efficiency of VSV by generating a strong bystander effect and by contributing to the activation of the immune system against the tumor without detrimentally altering the kinetics of virus-mediated oncolysis and may be useful in the treatment of malignant disease.

REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 11 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:620161 CAPLUS
DOCUMENT NUMBER: 139:244498
TITLE: Development of recombinant vesicular stomatitis viruses that exploit defects in host defense to augment specific oncolytic activity
AUTHOR(S): Obuchi, Masatsugu; Fernandez, Marilyn; Barber, Glen N.
CORPORATE SOURCE: Department of Microbiology and Immunology and Sylvester Comprehensive Cancer Center, University of Miami School of Medicine, Miami, FL, 33136, USA
SOURCE: Journal of Virology (2003), 77(16), 8843-8856
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Vesicular stomatitis virus (VSV) is a neg.-stranded RNA virus normally sensitive to the antiviral actions of alpha/beta interferon (IFN-.alpha./.beta.). Recently, the authors reported that VSV replicates to high levels in many transformed cells due, in part, to susceptible cells harboring defects in the IFN system. These observations were exploited to demonstrate that VSV can be used as a viral oncolytic agent to eradicate malignant cells in vivo while leaving normal tissue relatively unaffected. To attempt to improve the specificity and efficacy of this system as a potential tool in gene therapy and against malignant disease, the authors have genetically

engineered VSV that expresses the murine IFN-.beta. gene. The resultant virus (VSV-IFN.beta.) was successfully propagated in cells not receptive to murine IFN-.alpha./.beta. and expressed high levels of functional heterologous IFN-.beta.. In normal murine embryonic fibroblasts (MEFs), the growth of VSV-IFN.beta. was greatly reduced and diminished cytopathic effect was obsd. due to the prodn. of recombinant IFN-.beta., which by functioning in a manner involving autocrine and paracrine mechanisms induced an antiviral effect, preventing virus growth. However, VSV-IFN.beta. grew to high levels and induced the rapid apoptosis of transformed cells due to defective IFN pathways being prevalent and thus unable to initiate proficient IFN-mediated host defense. Importantly, VSV expressing the human IFN-.beta. gene (VSV-hIFN.beta.) behaved comparably and, while nonlytic to normal human cells, readily killed their malignant counterparts. Similar to the authors' in vitro observations, following i.v. and intranasal inoculation in mice, recombinant VSV (rVSV)-IFN.beta. was also significantly attenuated compared to wild-type VSV or rVSV expressing green fluorescent protein. However, VSV-IFN.beta. retained propitious oncolytic activity against metastatic lung disease in immunocompetent animals and was able to generate robust antitumor T-cell responses. The authors' data indicate that rVSV designed to exploit defects in mechanisms of host defense can provide the basis for new generations of effective, specific, and safer viral vectors for the treatment of malignant and other disease.

REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 12 OF 25 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:205204 CAPLUS

DOCUMENT NUMBER: 135:2622

TITLE: Oncolytic activity of vesicular stomatitis virus is effective against tumors exhibiting aberrant p53, Ras, or Myc function and involves the induction of apoptosis

AUTHOR(S): Balachandran, Siddharth; Porosnicu, Mercedes; Barber, Glen N.

CORPORATE SOURCE: Department of Microbiology and Immunology and Sylvester Comprehensive Cancer Center, University of Miami School of Medicine, Miami, FL, 33136, USA

SOURCE: Journal of Virology (2001), 75(7), 3474-3479
CODEN: JOVIAM; ISSN: 0022-538X

PUBLISHER: American Society for Microbiology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB We have recently shown that vesicular stomatitis virus (VSV)

exhibits potent oncolytic activity both in vitro and in vivo. In this study, we further demonstrated, in vivo, the efficacy of VSV antitumor action by showing that tumors that are defective in p53 function or transformed with myc or activated ras are also susceptible to viral cytolysis. The mechanism of viral oncolytic activity involved the induction of multiple caspase-dependent apoptotic pathways was effective in the absence of any significant cytotoxic T-lymphocyte response, and occurred despite normal PKR activity and eIF2.alpha. phosphorylation. In addn., VSV caused significant inhibition of tumor growth when administered i.v. in immunocompetent hosts. Our data indicate that VSV shows significant promise as an effective oncolytic agent against a wide variety of malignant diseases that harbor a diversity of genetic defects.

REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 13 OF 25 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2006:215422 BIOSIS

DOCUMENT NUMBER: PREV200600215554

TITLE: Oncolytic activity of vesicular stomatitis virus in primary adult T-cell leukemia.

AUTHOR(S): Cesaire, R.; Oliere, S.; Sharif-Askari, E.; Loignon, M.; Lezin, A.; Olindo, S.; Panelatti, G.; Kazanji, M.; Aloyz, R.; Panasci, L.; Bell, J. C.; Hiscott, J. [Reprint Author]

CORPORATE SOURCE: McGill Univ, Jewish Gen Hosp, Lady Davis Inst Med Res, Mol Oncol Grp, Room 526,3755 Cote Ste Catherine Rd, Montreal, PQ H3T 1E2, Canada
john.hiscott@mcgill.ca

SOURCE: Oncogene, (JAN 2006) Vol. 25, No. 3, pp. 349-358.
CODEN: ONCNES. ISSN: 0950-9232.

DOCUMENT TYPE: Article

LANGUAGE: English

ENTRY DATE: Entered STN: 29 Mar 2006
Last Updated on STN: 29 Mar 2006

AB Treatments for hematological malignancies have improved considerably over the past decade, but the growing therapeutic arsenal has not benefited adult T-cell leukemia (ATL) patients. Oncolytic viruses such as vesicular stomatitis virus (VSV) have recently emerged as a potential treatment of solid tumors and leukemias in vitro and in vivo. In the current study, we investigated the ability of VSV to lyse primary human T-lymphotropic virus type 1 (HTLV-1)-

infected T-lymphocytes from patients with ATL. Ex vivo primary ATL cells were permissive for VSV and underwent rapid oncolysis in a time-dependent manner. Importantly, VSV infection showed neither viral replication nor oncolysis in HTLV1- infected, nonleukemic cells from patients with HTLV-1associated myelopathy/ tropical spastic paraparesis (HAM/TSP), and in naive CD4+ T- lymphocytes from normal individuals or in ex vivo cell samples from patients with chronic lymphocytic leukemia (CLL). Interestingly, activation of primary CD4+ T- lymphocytes with antiCD3/ CD28 monoclonal antibody, and specifically with anti-CD3, was sufficient to induce limited viral replication and oncolysis. However, at a similar level of T- cell activation, VSV replication was increased fourfold in ATL cells compared to activated CD4+ T- lymphocytes, emphasizing the concept that VSV targets genetic defects unique to tumor cells to facilitate its replication. In conclusion, our findings provide the first essential information for the development of a VSV-based treatment for ATL.

L7 ANSWER 14 OF 25 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2005:290643 BIOSIS
DOCUMENT NUMBER: PREV200510081946
TITLE: Targeting human glioblastoma cells: Comparison of nine viruses with oncolytic potential.
AUTHOR(S): Wollmann, Guido; Tattersall, Peter; van den Pol, Anthony N. [Reprint Author]
CORPORATE SOURCE: Yale Univ, Sch Med, Dept Neurosurg, 333 Cedar St, New Haven, CT 06520 USA
anthony.vandenpol@yale.edu
SOURCE: Journal of Virology, (MAY 2005) Vol. 79, No. 10, pp. 6005-6022.
CODEN: JOVIAM. ISSN: 0022-538X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Aug 2005
Last Updated on STN: 4 Aug 2005
AB Brain tumors classified as glioblastomas have proven refractory to treatment and generally result in death within a year of diagnosis. We used seven in vitro tests and one in vivo trial to compare the efficacy of nine different viruses for targeting human glioblastoma. Green fluorescent protein (GFP)-expressing vesicular stomatitis (VSV), Sindbis virus, pseudorabies virus (PRV), adeno-associated virus (AAV), and minute virus of mice i-strain (MVMi) and MVMp all infected glioblastoma cells. Mouse and human cytomegalovirus, and simian virus 40 showed only low levels of infection or GFP expression.

VSV and Sindbis virus showed strong cytolytic actions and high rates of replication and spread, leading to an elimination of glioblastoma. PRV and both MVM strains generated more modest lytic effects and replication capacity. VSV showed a similar oncolytic profile on U-87 MG and M059J glioblastoma. In contrast, Sindbis virus showed strong preference for U-87 MG, whereas MVMi and MVMp preferred M059J. Sindbis virus and both MVM strains showed highly tumor-selective actions in glioblastoma plus fibroblast coculture. VSV and Sindbis virus were serially passaged on glioblastoma cells; we isolated a variant, VSV-rp30, that had increased selectivity and lytic capacity in glioblastoma cells. VSV and Sindbis virus were very effective at replicating, spreading within, and selectively killing human glioblastoma in an in vivo mouse model, whereas PRV and AAV remained at the injection site with minimal spread. Together, these data suggest that four (VSV, Sindbis virus, MVMi, and MVMp) of the nine viruses studied merit further analysis for potential therapeutic actions on glioblastoma.

L7 ANSWER 15 OF 25 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2005:82408 BIOSIS
DOCUMENT NUMBER: PREV200500076460
TITLE: Sensitivity of prostate tumors to wild type and M protein mutant vesicular stomatitis viruses.
AUTHOR(S): Ahmed, Maryam [Reprint Author]; Cramer, Scott D.; Lyles, Douglas S.
CORPORATE SOURCE: Sch MedDept Biochem, Wake Forest Univ, Med Ctr Blvd, Winston Salem, NC, 27157, USA
mahmed@wfubmc.edu
SOURCE: Virology, (December 5 2004) Vol. 330, No. 1, pp. 34-49. print.
ISSN: 0042-6822 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Feb 2005
Last Updated on STN: 23 Feb 2005

AB Because of its potent ability to induce apoptosis, vesicular stomatitis virus (VSV) is an attractive candidate as an oncolytic virus for tumor therapy. Previous studies have suggested that VSV selectively infects tumor cells due to defects in their antiviral responses making them more susceptible to VSV infection than normal cells. We tested this hypothesis in the prostate tumor system by comparing LNCaP and PC-3 prostate tumor cells to benign human prostatic epithelial cells from patient prostatectomy specimens. We compared the cell killing ability of a recombinant virus containing a wild-type (wt) M

protein (rwt) and an isogenic M protein mutant virus (rM51R-M) that induces interferon (IFN) in infected cells and should display a greater selectivity for tumor cells. Our results showed that in single-cycle infection experiments, LNCaP cells were sensitive to killing by both wt and mutant viruses, while PC-3 cells were highly resistant to VSV -induced cell killing. LNCaP and benign prostate cells were similarly susceptible to both viruses, indicating that normal prostate cells are not inherently resistant to killing by VSV. In each of the cell lines, the rM51R-M virus induced similar levels of apoptosis to rwt virus, showing that the M protein does not play a significant role in apoptosis induction by VSV in these cells. In multiple-cycle infection experiments, LNCaP cells were more sensitive than benign prostatic epithelial cells to virus-induced cell killing by rM51R-M virus, but not rwt virus. Both viruses were equally effective at reducing LNCaP tumor volume in vivo following intratumoral and intravenous inoculation in nude mice, while PC-3 tumors were resistant to VSV treatment. None of the mice treated with rM51R-M virus died as a result of virus infection, while 50-71% of mice treated with rwt virus succumbed to virus infection. Similarly, when inoculated by the more sensitive intranasal route, the rM51R-M virus was less pathogenic than the rwt virus from which it was derived. These results indicate that M protein mutant viruses are superior candidates as oncolytic viruses for therapies of prostate tumors, but future strategies for use of VSV will require testing individual tumors for their susceptibility to virus infection.

L7 ANSWER 16 OF 25 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2004:463465 BIOSIS
DOCUMENT NUMBER: PREV200400464964
TITLE: Induction of apoptosis and tumor regression by vesicular stomatitis virus in the presence of gemcitabine in lung cancer.
AUTHOR(S): Li, Qiu; Wei, Yu-quan [Reprint Author]; Wen, Yan-jun; Zhao, Xia; Tian, Ling; Yang, Li; Mao, Yong-qiu; Kan, Bing; Wu, Yang; Ding, Zhen-yu; Deng, Hong-Xin; Li, Jiong; Luo, Yan; Li, Hong-; He, Qiu-ming; Su, Jing-mei; Xiao, Fei; Zou, Chun-Hua; Fu, Chun-Hua; Xie, Xing-jiang; Yi, Tao; Tan, Guang-Hong; Wang, Lian; Chen, Jing; Liu, Jian; Gao, Zhen-Nan
CORPORATE SOURCE: W China Med SchW China HospLab Biotherapy Human Dis,Minist
Educ, Sichuan Univ, Guo Xue Xiang 37, Chengdu, Sichuan, 610041, China
yuquanwei@vip.sina.com

SOURCE: International Journal of Cancer, (October 20 2004) Vol. 112, No. 1, pp. 143-149. print.
CODEN: IJCNAW. ISSN: 0020-7136.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Dec 2004
Last Updated on STN: 1 Dec 2004

AB Vesicular stomatitis virus (VSV) has been shown to replicate rapidly in vitro and kill selectively a variety of tumor cell lines. The present study was designed to determine whether gemcitabine potentiates the antitumor activity of VSV in vitro and in vivo.
A549 human lung adenocarcinoma cells and LLC Lewis lung carcinoma cells were treated with VSV (0.1-10 plaque-forming units per cell) plus gemcitabine (20 nM to 20 muM). Mice bearing A549 or LLC were treated with VSV (5 x 104 to 1 x 108 plaque-forming units) daily for 5 days plus gemcitabine (5-125 mg/kg/day) once every 3 days for 4 times. Induction of apoptosis and effects on growth inhibition were assessed. The lung cancer cells treated with VSV plus gemcitabine displayed the apparently increased apoptotic cells compared with treatment with VSV or gemcitabine alone. The combined treatment with VSV plus gemcitabine induced the apparent antitumor activity with complete regression of the established lung cancer in both A549 and LLC lung cancer models and augmented the induction of apoptosis in lung cancer cells in vivo as well. This study suggests that the combined treatment with VSV plus gemcitabine may augment the induction of apoptosis in lung cancer cells in vitro and in vivo, and that the augmented antitumor activity in vivo may result from the increased induction of apoptosis in lung cancer cells. The present findings may be of importance to the further exploration of the potential application of this combined approach in the treatment of lung cancer.

L7 ANSWER 17 OF 25 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:450979 BIOSIS
DOCUMENT NUMBER: PREV200400452923
TITLE: Replication and cytopathic effect of oncolytic vesicular stomatitis virus in hypoxic tumor cells in vitro and in vivo.
AUTHOR(S): Connor, John H. [Reprint Author]; Naczki, Christine; Koumenis, Costas; Lyles, Douglas S.
CORPORATE SOURCE: Sch MedDept Microbiol and Immunol, Wake Forest Univ, Med Ctr Blvd, Winston Salem, NC, 27157, USA
jconnor@wfubmc.edu

SOURCE: Journal of Virology, (September 2004) Vol. 78, No. 17, pp. 8960-8970. print.
ISSN: 0022-538X (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Nov 2004
Last Updated on STN: 24 Nov 2004

AB Tumor hypoxia presents an obstacle to the effectiveness of most antitumor therapies, including treatment with oncolytic viruses. In particular, an oncolytic virus must be resistant to the inhibition of DNA, RNA, and protein synthesis that occurs during hypoxic stress. Here we show that vesicular stomatitis virus (VSV), an oncolytic RNA virus, is capable of replication under hypoxic conditions. In cells undergoing hypoxic stress, VSV infection produced larger amounts of mRNA than under normoxic conditions. However, translation of these mRNAs was reduced at earlier times postinfection in hypoxia-adapted cells than in normoxic cells. At later times postinfection, VSV overcame a hypoxia-associated increase in ox subunit of eukaryotic initiation factor 2 (eIF-2alpha) phosphorylation and initial suppression of viral protein synthesis in hypoxic cells to produce large amounts of viral protein. VSV infection caused the dephosphorylation of the translation initiation factor eIF-4E and inhibited host translation similarly under both normoxic and hypoxic conditions. VSV produced progeny virus to similar levels in hypoxic and normoxic cells and showed the ability to expand from an initial infection of 1% of hypoxic cells to spread through an entire population. In all cases, virus infection induced classical cytopathic effects and apoptotic cell death. When VSV was used to treat tumors established in nude mice, we found VSV replication in hypoxic areas of these tumors. This occurred whether the virus was administered intratumorally or intravenously. These results show for the first time that VSV has an inherent capacity for infecting and killing hypoxic cancer cells. This ability could represent a critical advantage over existing therapies in treating established tumors.

L7 ANSWER 18 OF 25 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:423955 BIOSIS
DOCUMENT NUMBER: PREV200400419862
TITLE: Vesicular stomatitis virus: A potential therapeutic virus for the treatment of hematologic malignancy.
AUTHOR(S): Lichty, Brian D.; Stojdl, David F.; Taylor, Rebecca A.; Miller, Leigh; Frenkel, Irina; Atkins, Harold; Bell, John C. [Reprint Author]
CORPORATE SOURCE: Res Labs, Ottawa Reg Canc Ctr, 503 Smyth Rd, Ottawa, ON,

K1H 1C4, Canada
jbell@ohri.ca
SOURCE: Human Gene Therapy, (September 2004) Vol. 15, No. 9, pp. 821-831. print.
ISSN: 1043-0342 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 3 Nov 2004
Last Updated on STN: 3 Nov 2004
AB Certain strains of vesicular stomatitis virus (VSV) have been shown to be oncolytic in a wide variety of solid tumors. In the present study, we tested the leukemolytic properties of VSV using established leukemia cell lines and primary patient material. VSV efficiently killed essentially all leukemic cell lines. In contrast, however, normal clonogenic bone marrow progenitor cells and peripheral blood cells were remarkably refractory to infection by VSV. By exploiting this large difference in susceptibility to infection we successfully purged contaminating leukemic cells from cultures of peripheral blood progenitor cells (PBPC) using VSV. VSV was also able to infect and kill leukemic cells in primary samples taken from patients with multiple myeloma (MM). This study demonstrates the potential utility of VSV in the treatment, both ex vivo and in vivo, of hematologic malignancies.

L7 ANSWER 19 OF 25 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:293987 BIOSIS
DOCUMENT NUMBER: PREV200400292339
TITLE: Syncytia induction enhances the oncolytic potential of vesicular stomatitis virus in virotherapy for cancer.
AUTHOR(S): Ebert, Oliver; Shinozaki, Katsunori; Kournioti, Chryssanthi; Park, Man-Seong; Garcia-Sastre, Adolfo; Woo, Savio L. C. [Reprint Author]
CORPORATE SOURCE: Carl C Icahn Ctr Gene Therapy and Mol Med, Mt Sinai Sch Med, 1 Gustave L Levy Pl,Box 1496, New York, NY, 10029, USA
savio.woo@mssm.edu
SOURCE: Cancer Research, (May 1 2004) Vol. 64, No. 9, pp. 3265-3270. print.
ISSN: 0008-5472 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Jun 2004
Last Updated on STN: 23 Jun 2004
AB Vesicular stomatitis virus; (VSV) selectively replicates in

tumor but not in normal cells and is being developed as an oncolytic agent for cancer therapy. Here we report the construction of a recombinant VSV capable of inducing syncytia formation between tumor cells through membrane fusion at neutral pH, which led to enhanced oncolytic properties against multifocal hepatocellular carcinoma (HCC) in the livers of immunocompetent rats. Recombinant VSV vectors were constructed by insertion into their genome a transcription unit expressing a control or fusion protein derived from Newcastle disease virus. In vitro characterization of the recombinant fusogenic VSV vector on human and rat HCC cells showed extensive syncytia formation and significantly enhanced cytotoxic effects. lit vivo, administration of fusogenic VSV into the hepatic artery of Buffalo rats bearing syngeneic multifocal HCC lesions in their livers resulted in syncytia formation exclusively within the tumors, and there was no collateral damage to the neighboring hepatic parenchyma. The fusogenic VSV also conferred a significant survival advantage over a nonfusogenic control virus in the treated animals (P = 0.0078, log-rank test). The results suggest that fusogenic VSV can be developed into an effective and safe therapeutic agent for cancer treatment in patients, including those with multifocal HCC in the liver.

L7 ANSWER 20 OF 25 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2004:204281 BIOSIS
DOCUMENT NUMBER: PREV200400204824
TITLE: Using a novel organotypic brain slice - glioma CO - culture model in studying recombinant VSV vectors as oncolytic agents.
AUTHOR(S): Zhou, Q. [Reprint Author]; Duntsch, C. D. [Reprint Author]; Jayakar, H. R.; Weimar, J. D. [Reprint Author]; Whitt, M. A.
CORPORATE SOURCE: Dept. of Neurosurgery, The Univ. Tennessee Hlth. Sci. Ctr., Memphis, TN, USA
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 752.15. http://sfn.scholarone.com. e-file.
Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004

AB We have developed an in vitro organotypic brain slice-glioma co-culture model to evaluate vesicular stomatitis virus (VSV) vectors as potential oncolytic therapeutic agents for high-grade gliomas. The brain slices of post-natal day 1-2 rats were used to develop the slice culture. Cortex, striatum, and substantia nigra were dissected out and placed on a porous membrane on coverslips. The slices were cultured at 35degreeC for 2 wk allowing neurons to develop synaptic connections similar to that found in vivo. The C6-GFP glioma cells resuspended in methylcellulose were then introduced into the culture resulting in a tumor cluster 2-3 d after inoculation. Using a recombinant, wild-type, VSV (rVSV-wt), we found that C6 glioma cells were rapidly eliminated from the co-culture, but with significant damage to neurons as evaluated by loss of MAP-2 immunoreactivity. However, pretreatment with interferon-beta (IFNbeta) virtually eliminated VSV infection of normal tissue without reducing the oncolytic effects on tumor cells. But IFNbeta pretreatment could not prevent normal tissue from damaging when exposed to rVSV-wt. In contrast, a replication-restricted vector that has the glycoprotein gene deleted (rVSV-DELTAG), in combination with IFNbeta pretreatment, could effectively eliminate glioma cells growth in the slice without causing significant infection or damage to the cells of normal tissue in the co-culture. In summary, this co-culture model is a powerful tool for therapeutic studies of the CNS that allows studies of normal tissue toxicity and tumor lytic efficacy simultaneously. These in vitro studies provide that rVSV-DELTAG is an effective oncolytic agent with minimal toxicity and therefore, could be considered for development as an adjuvant to surgery in the treatment of glioma.

L7 ANSWER 21 OF 25 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:47308 BIOSIS

DOCUMENT NUMBER: PREV200400039977

TITLE: The oncolytic effect of recombinant vesicular stomatitis virus is enhanced by expression of the fusion cytosine deaminase/uracil phosphoribosyltransferase suicide gene.

AUTHOR(S): Porosnicu, Mercedes; Mian, Abdul; Barber, Glen N. [Reprint Author]

CORPORATE SOURCE: University of Miami School of Medicine, 1550 North West 10th Avenue, Room 514, Papanicolaou Building, M710, Miami, FL, 33136, USA
gbarber@med.miami.edu

SOURCE: Cancer Research, (December 1 2003) Vol. 63, No. 23, pp. 8366-8376. print.
ISSN: 0008-5472 (ISSN print).

DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Jan 2004
Last Updated on STN: 14 Jan 2004

AB Vesicular stomatitis virus (VSV) has recently been demonstrated to exhibit significant oncolytic capabilities against a wide variety of tumor models in vitro and in vivo. To potentially enhance the oncolytic effect, we generated a novel recombinant VSV (rVSV) that expressed the fusion suicide gene Escherichia coli cytosine deaminase (CD)/uracil phosphoribosyltransferase (UPRT). rVSV encoding the CD/UPRT fusion gene (VSV-C:U) exhibited normal growth properties and generated high levels of biologically active CD/UPRT that could catalyze the modification of 5-fluorocytosine into chemotherapeutic 5-fluorouracil (5-FU), which exhibited considerable bystander effect. Intratumoral inoculation of VSV-C:U in the presence of the systemically administered prodrug 5-fluorocytosine produced statistically significant reductions in the malignant growth of syngeneic lymphoma (A20) or mammary carcinoma (TSA) in BALB/c mice compared with rVSV treatments or with control 5-FU alone. Aside from detecting prolonged therapeutic levels of 5-FU in VSV -C:U-treated animals harboring TSA tumors and enhancing bystander killing of tumor cells, we demonstrated marked activation of IFN-gamma-secreting cytotoxic T cells by enzyme-linked immunospot analysis that may have also facilitated tumor killing. In conclusion, the insertion of the fusion CD/UPRT suicide gene potentiates the oncolytic efficiency of VSV by generating a strong bystander effect and by contributing to the activation of the immune system against the tumor without detrimentally altering the kinetics of virus-mediated oncolysis and may be useful in the treatment of malignant disease.

L7 ANSWER 22 OF 25 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:432731 BIOSIS
DOCUMENT NUMBER: PREV200300432731
TITLE: Development of recombinant vesicular stomatitis viruses that exploit defects in host defense to augment specific oncolytic activity.
AUTHOR(S): Obuchi, Masatsugu; Fernandez, Marilyn; Barber, Glen N. [Reprint Author]
CORPORATE SOURCE: University of Miami School of Medicine, 1550 NW 10th Ave., Rm. 511, Papanicolaou Building, M710, Miami, FL, 33136, USA gbarber@med.miami.edu
SOURCE: Journal of Virology, (August 2003) Vol. 77, No. 16, pp. 8843-8856. print.

ISSN: 0022-538X (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Sep 2003
Last Updated on STN: 17 Sep 2003
AB Vesicular stomatitis virus (VSV) is a negative-stranded RNA virus normally sensitive to the antiviral actions of alpha/beta interferon (IFN-alpha/beta). Recently, we reported that VSV replicates to high levels in many transformed cells due, in part, to susceptible cells harboring defects in the IFN system. These observations were exploited to demonstrate that VSV can be used as a viral oncolytic agent to eradicate malignant cells in vivo while leaving normal tissue relatively unaffected. To attempt to improve the specificity and efficacy of this system as a potential tool in gene therapy and against malignant disease, we have genetically engineered VSV that expresses the murine IFN-beta gene. The resultant virus (VSV -IFNbeta) was successfully propagated in cells not receptive to murine IFN-alpha/beta and expressed high levels of functional heterologous IFN-beta. In normal murine embryonic fibroblasts (MEFs), the growth of VSV-IFNbeta was greatly reduced and diminished cytopathic effect was observed due to the production of recombinant IFN-beta, which by functioning in a manner involving autocrine and paracrine mechanisms induced an antiviral effect, preventing virus growth. However, VSV-IFNbeta grew to high levels and induced the rapid apoptosis of transformed cells due to defective IFN pathways being prevalent and thus unable to initiate proficient IFN-mediated host defense. Importantly, VSV expressing the human IFN-beta gene (VSV-hIFNbeta) behaved comparably and, while nonlytic to normal human cells, readily killed their malignant counterparts. Similar to our in vitro observations, following intravenous and intranasal inoculation in mice, recombinant VSV (rVSV)-IFNbeta was also significantly attenuated compared to wild-type VSV or rVSV expressing green fluorescent protein. However, VSV-IFNbeta retained propitious oncolytic activity against metastatic lung disease in immunocompetent animals and was able to generate robust antitumor T-cell responses. Our data indicate that rVSV designed to exploit defects in mechanisms of host defense can provide the basis for new generations of effective, specific, and safer viral vectors for the treatment of malignant and other disease.

L7 ANSWER 23 OF 25 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:215362 BIOSIS
DOCUMENT NUMBER: PREV200200215362
TITLE: Cytolytic viruses as potential anti-cancer agents.

AUTHOR(S): Ring, Christopher J. A. [Reprint author]
CORPORATE SOURCE: Gene Interference, Medicines Research Centre, Glaxo SmithKline Research and Development, Gunnels Wood Road, Stevenage, Herts, SG1 2NY, UK
cjr48991@gsk.com
SOURCE: Journal of General Virology, (March, 2002) Vol. 83, No. 3, pp. 491-502. print.
CODEN: JGVIAY. ISSN: 0022-1317.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Mar 2002
Last Updated on STN: 27 Mar 2002
AB The resistance of cancers to conventional therapies has inspired the search for novel strategies. One such approach, namely gene therapy, is based upon the introduction of genes such as those encoding suicide proteins, tumour suppressor proteins or cytokines into tumour cells by means of a genetic vector. The efficiency with which viruses transfer their genes from one host cell to another has led to the widespread use of viruses as genetic vectors. For safety reasons, such virus vectors are generally replication-defective but, unfortunately, this has limited the efficacy of treatment by restricting the number of cells to which the therapeutic gene is delivered. For this reason, the use of replication-competent viruses has been proposed, since virus replication would be expected to lead to amplification and spread of the therapeutic genes in vivo. The replication of many viruses results in lysis of the host cells. This inherent cytotoxicity, together with the efficiency with which viruses can spread from one cell to another, has inspired the notion that replication-competent viruses could be exploited for cancer treatment. Some viruses have been shown to replicate more efficiently in transformed cells but it is unlikely that such examples will exhibit a high enough degree of tumour selectivity, and hence safety, for the treatment of patients. Our increasing knowledge of the pathogenesis of virus disease and the ability to manipulate specific regions of viral genomes have allowed the construction of viruses that are attenuated in normal cells but retain their ability to lyse tumour cells. Such manipulations have included modifying the ability of viruses to bind to, or replicate in, particular cell types, while others have involved the construction of replication-competent viruses encoding suicide proteins or cytokines. Naturally occurring or genetically engineered oncolytic viruses based upon adenovirus, herpes simplex virus, Newcastle disease virus, poliovirus, vesicular stomatitis virus, weasles virus and reovirus have been described. The results of animal studies are encouraging and a number of viruses are now being evaluated in clinical trials.

L7 ANSWER 24 OF 25 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2001:242607 BIOSIS
DOCUMENT NUMBER: PREV200100242607
TITLE: Oncolytic activity of vesicular stomatitis virus is effective against tumors exhibiting aberrant p53, ras, or myc function and involves the induction of apoptosis.
AUTHOR(S): Balachandran, Siddharth; Porosnicu, Mercedes; Barber, Glen N. [Reprint author]
CORPORATE SOURCE: University of Miami School of Medicine, 1550 NW 10th Ave., Rm. 514, Papanicolaou Building, M710, Miami, FL, 33136, USA gbarber@med.miami.edu
SOURCE: Journal of Virology, (April, 2001) Vol. 75, No. 7, pp. 3474-3479. print.
CODEN: JOVIAM. ISSN: 0022-538X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 16 May 2001
Last Updated on STN: 19 Feb 2002
AB We have recently shown that vesicular stomatitis virus (VSV) exhibits potent oncolytic activity both in vitro and in vivo (S. Balachandran and G. N. Barber, IUBMB Life 50:135-138, 2000). In this study, we further demonstrated, in vivo, the efficacy of VSV antitumor action by showing that tumors that are defective in p53 function or transformed with myc or activated ras are also susceptible to viral cytolysis. The mechanism of viral oncolytic activity involved the induction of multiple caspase-dependent apoptotic pathways was effective in the absence of any significant cytotoxic T-lymphocyte response, and occurred despite normal PKR activity and eIF2alpha phosphorylation. In addition, VSV caused significant inhibition of tumor growth when administered intravenously in immunocompetent hosts. Our data indicate that VSV shows significant promise as an effective oncolytic agent against a wide variety of malignant diseases that harbor a diversity of genetic defects.

L7 ANSWER 25 OF 25 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2001:53684 BIOSIS
DOCUMENT NUMBER: PREV200100053684
TITLE: Vesicular stomatitis virus (VSV) therapy of tumors.
AUTHOR(S): Balachandran, Siddharth; Barber, Glen N. [Reprint author]

CORPORATE SOURCE: University of Miami School of Medicine, 1550 NW 10th Ave.,
514 Papanicolaou Bldg., Miami, FL, 33136, USA
gbarber@med.miami.edu
SOURCE: IUBMB Life, (August, 2000) Vol. 50, No. 2, pp. 135-138.
print.
ISSN: 1521-6543.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Jan 2001
Last Updated on STN: 12 Feb 2002
AB Vesicular stomatitis virus (VSV) is an essentially nonpathogenic negative-stranded RNA virus, the replication of which is extremely sensitive to the antiviral effects of interferon (IFN). We demonstrate here that VSV selectively induces the cytolysis of numerous transformed human cell lines in vitro, with all the morphological characteristics of apoptotic cell death. Importantly, VSV can also potently inhibit the growth of p53-null C6 glioblastoma tumors in vivo without infecting and replicating in normal tissue. With our previous findings demonstrating that primary cells containing the double-stranded RNA-activated protein kinase PKR and a functional IFN system are not permissive to VSV replication, these results suggest that signaling by IFN may be defective in many malignancies. Thus VSV might be useful in novel therapeutic strategies for targeting neoplastic disease.

=> D L3 IBIB TI 1-58

L3 ANSWER 1 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2006:198241 CAPLUS
DOCUMENT NUMBER: 144:346651
TITLE: Matrix protein mutant of vesicular stomatitis virus stimulates maturation of myeloid dendritic cells
AUTHOR(S): Ahmed, Maryam; Brzoza, Kristina L.; Hiltbold, Elizabeth M.
CORPORATE SOURCE: Department of Biochemistry, Wake Forest University School of Medicine, Winston-Salem, NC, 27157, USA
SOURCE: Journal of Virology (2006), 80(5), 2194-2205
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Matrix protein mutant of vesicular stomatitis virus stimulates maturation of myeloid dendritic cells

REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2006:52327 CAPLUS
DOCUMENT NUMBER: 144:425241
TITLE: Oncolytic activity of vesicular stomatitis virus in primary adult T-cell leukemia
AUTHOR(S): Cesaire, R.; Oliere, S.; Sharif-Askari, E.; Loignon, M.; Lezin, A.; Olindo, S.; Panelatti, G.; Kazanji, M.; Aloyz, R.; Panasci, L.; Bell, J. C.; Hiscott, J.
CORPORATE SOURCE: Laboratoire de Virologie-Immunologie and, Centre Hospitalier Universitaire de Fort-de-France, Martinique, Fr.
SOURCE: Oncogene (2006), 25(3), 349-358
CODEN: ONCNES; ISSN: 0950-9232
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Oncolytic activity of vesicular stomatitis virus in primary adult T-cell leukemia
REFERENCE COUNT: 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1229042 CAPLUS
DOCUMENT NUMBER: 144:49701
TITLE: VSV-tumor selective replication and protein translation
AUTHOR(S): Barber, Glen N.
CORPORATE SOURCE: Department of Microbiology and Immunology, Sylvester Comprehensive Cancer Center, University of Miami School of Medicine, Miami, FL, 33136, USA
SOURCE: Oncogene (2005), 24(52), 7710-7719
CODEN: ONCNES; ISSN: 0950-9232
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
TI VSV-tumor selective replication and protein translation
REFERENCE COUNT: 92 THERE ARE 92 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:1170417 CAPLUS
DOCUMENT NUMBER: 143:420671
TITLE: Prophylactic alpha interferon treatment increases the therapeutic index of oncolytic vesicular stomatitis virus virotherapy for advanced hepatocellular carcinoma in immune-competent rats
AUTHOR(S): Shinozaki, Katsunori; Ebert, Oliver; Suriawinata, Arief; Thung, Swan N.; Woo, Savio L. C.
CORPORATE SOURCE: Department of Gene and Cell Medicine, Mount Sinai School of Medicine, New York, NY, 10029-6574, USA
SOURCE: Journal of Virology (2005), 79(21), 13705-13713
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Prophylactic alpha interferon treatment increases the therapeutic index of oncolytic vesicular stomatitis virus virotherapy for advanced hepatocellular carcinoma in immune-competent rats
REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 5 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1038076 CAPLUS
DOCUMENT NUMBER: 144:80330
TITLE: Vesicular stomatitis: an oncolytic virus that exploits tumor-specific defects in the interferon pathway
AUTHOR(S): Taylor, Rebecca Ann C.; Paterson, Jennifer M.; Bell, John C.
CORPORATE SOURCE: Research Laboratories, Ottawa Regional Cancer Centre, Ottawa, ON, Can.
SOURCE: Viral Therapy of Human Cancers (2005), 597-625.
Editor(s): Sinkovics, Joseph G.; Horvath, Joseph C.
Marcel Dekker, Inc.: New York, N. Y.
CODEN: 69HIM6; ISBN: 0-8247-5913-3
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English
TI Vesicular stomatitis: an oncolytic virus that exploits tumor-specific defects in the interferon pathway
REFERENCE COUNT: 84 THERE ARE 84 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 6 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:421155 CAPLUS

DOCUMENT NUMBER: 143:93884
TITLE: Targeting human glioblastoma cells: Comparison of nine viruses with oncolytic potential
AUTHOR(S): Wollmann, Guido; Tattersall, Peter; van den Pol, Anthony N.
CORPORATE SOURCE: Department of Neurosurgery, Yale University School of Medicine, New Haven, CT, 06520, USA
SOURCE: Journal of Virology (2005), 79(10), 6005-6022
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Targeting human glioblastoma cells: Comparison of nine viruses with oncolytic potential
REFERENCE COUNT: 67 THERE ARE 67 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 7 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:241128 CAPLUS
DOCUMENT NUMBER: 142:423269
TITLE: Treatment of multi-focal colorectal carcinoma metastatic to the liver of immune-competent and syngeneic rats by hepatic artery infusion of oncolytic vesicular stomatitis virus
AUTHOR(S): Shinozaki, Katsunori; Ebert, Oliver; Woo, Savio L. C.
CORPORATE SOURCE: Department of Gene and Cell Medicine, Mount Sinai School of Medicine, New York, NY, 10029-6574, USA
SOURCE: International Journal of Cancer (2005), 114(4), 659-664
CODEN: IJCNAW; ISSN: 0020-7136
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Treatment of multi-focal colorectal carcinoma metastatic to the liver of immune-competent and syngeneic rats by hepatic artery infusion of oncolytic vesicular stomatitis virus
REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 8 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:228828 CAPLUS
DOCUMENT NUMBER: 142:366996
TITLE: Systemic therapy of experimental breast cancer metastases by mutant vesicular stomatitis virus in

immune-competent mice
AUTHOR(S): Ebert, Oliver; Harbaran, Sonal; Shinozaki, Katsunori; Woo, Savio L. C.
CORPORATE SOURCE: Department of Gene and Cell Medicine, Mount Sinai School of Medicine, New York, NY, USA
SOURCE: Cancer Gene Therapy (2005), 12(4), 350-358
CODEN: CGTHEG; ISSN: 0929-1903
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Systemic therapy of experimental breast cancer metastases by mutant vesicular stomatitis virus in immune-competent mice
REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 9 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:25741 CAPLUS
DOCUMENT NUMBER: 142:132506
TITLE: Vesicular stomatitis virus as an oncolytic vector
AUTHOR(S): Barber, Glen N.
CORPORATE SOURCE: Department of Microbiology and Immunology, Sylvester Comprehensive Cancer Center, University of Miami School of Medicine, Miami, FL, USA
SOURCE: Viral Immunology (2004), 17(4), 516-527
CODEN: VIIMET; ISSN: 0882-8245
PUBLISHER: Mary Ann Liebert, Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
TI Vesicular stomatitis virus as an oncolytic vector
REFERENCE COUNT: 95 THERE ARE 95 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 10 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:953474 CAPLUS
TITLE: Sensitivity of prostate tumors to wild type and M protein mutant vesicular stomatitis viruses
AUTHOR(S): Ahmed, Maryam; Cramer, Scott D.; Lyles, Douglas S.
CORPORATE SOURCE: Department of Biochemistry, Wake Forest University School of Medicine, Winston-Salem, NC, 27157, USA
SOURCE: Virology (2004), 330(1), 34-49
CODEN: VIRLAX; ISSN: 0042-6822
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal

LANGUAGE: English
TI Sensitivity of prostate tumors to wild type and M protein mutant vesicular stomatitis viruses
REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 11 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:824043 CAPLUS
DOCUMENT NUMBER: 141:325690
TITLE: VSV mutants contg. mutations in matrix protein capable of stimulating cytokine prodn. and shutting down innate immunity and use thereof as vaccines and anti-cancer agents
INVENTOR(S): Bell, John C.; Lichty, Brian D.; Stojdl, David F.
PATENT ASSIGNEE(S): Ottawa Health Research Institute, Can.; Wellstat Biologics Corporation
SOURCE: PCT Int. Appl., 93 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004085659	A2	20041007	WO 2004-CA463	20040329
WO 2004085659	A3	20041209		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CN 1788088	A	20060614	CN 2004-80008416	20040329

PRIORITY APPLN. INFO.: US 2003-457591P P 20030327
TI VSV mutants contg. mutations in matrix protein capable of stimulating cytokine prodn. and shutting down innate immunity and use thereof as vaccines and anti-cancer agents

L3 ANSWER 12 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:824042 CAPLUS
DOCUMENT NUMBER: 141:325689
TITLE: Mutant vesicular stomatitis viruses containing mutations in matrix protein capable of stimulating cytokine production and shutting down innate immunity and use thereof as vaccines and anti-cancer agents
INVENTOR(S): Bell, John C.; Lichty, Brian D.; Stojdl, David F.
PATENT ASSIGNEE(S): Ottawa Health Research Institute, Can.; Wellstat Biologics Corporation
SOURCE: PCT Int. Appl., 106 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004085658	A1	20041007	WO 2004-CA460	20040329
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004223808	A1	20041007	AU 2004-223808	20040329
CA 2520279	AA	20041007	CA 2004-2520279	20040329
EP 1606411	A1	20051221	EP 2004-723949	20040329
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK				
CN 1788088	A	20060614	CN 2004-80008416	20040329

PRIORITY APPLN. INFO.: US 2003-457591P P 20030327
WO 2004-CA460 W 20040329

TI Mutant vesicular stomatitis viruses containing mutations in matrix protein capable of stimulating cytokine production and shutting down innate immunity and use thereof as vaccines and anti-cancer agents

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 13 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:725783 CAPLUS
TITLE: Vesicular Stomatitis Virus: A Potential Therapeutic Virus for the Treatment of Hematologic Malignancy
AUTHOR(S): Lichty, Brian D.; Stojdl, David F.; Taylor, Rebecca A.; Miller, Leigh; Frenkel, Irina; Atkins, Harold; Bell, John C.
CORPORATE SOURCE: Ottawa Regional Cancer Centre Research Laboratories, Ottawa, ON, K1H 1C4, Can.
SOURCE: Human Gene Therapy (2004), 15(9), 821-831
CODEN: HGTHE3; ISSN: 1043-0342
PUBLISHER: Mary Ann Liebert, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Vesicular Stomatitis Virus: A Potential Therapeutic Virus for the Treatment of Hematologic Malignancy
REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 14 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:723238 CAPLUS
TITLE: Oncolytic virus
INVENTOR(S): Bell, John C.; Sonenberg, Nahum; Stojdl, David F.; Brown, Earl G.; Atkins, Harold L.; Marius, Ricardo M.; Lichty, Brian D.; Knowles, Shane B.
PATENT ASSIGNEE(S): Can.
SOURCE: U.S. Pat. Appl. Publ., Division of Ser. No. US 2000-664444, filed on 18 Sep 2000 which
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004170607	A1	20040902	US 2003-743649	20031223
US 2004208849	A1	20041021	US 2003-743639	20031222

PRIORITY APPLN. INFO.: US 1999-287590P P 19990917
US 2000-664444 A3 20000918
TI Oncolytic virus

L3 ANSWER 15 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:706181 CAPLUS
DOCUMENT NUMBER: 141:346449
TITLE: Replication and cytopathic effect of oncolytic

vesicular stomatitis virus in hypoxic tumor cells in vitro and in vivo
AUTHOR(S): Connor, John H.; Naczki, Christine; Koumenis, Costas; Lyles, Douglas S.
CORPORATE SOURCE: Department of Biochemistry, Wake Forest University School of Medicine, Winston-Salem, NC, USA
SOURCE: Journal of Virology (2004), 78(17), 8960-8970
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Replication and cytopathic effect of oncolytic vesicular stomatitis virus in hypoxic tumor cells in vitro and in vivo
REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 16 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:534872 CAPLUS
DOCUMENT NUMBER: 142:16351
TITLE: Recombinant vesicular stomatitis virus vectors as oncolytic agents in the treatment of high-grade gliomas in an organotypic brain tissue slice-glioma coculture model
AUTHOR(S): Duntsch, Christopher D.; Zhou, Qihong; Jayakar, Himangi R.; Weimar, James D.; Robertson, Jon H.; Pfeffer, Lawrence M.; Wang, Lie; Xiang, Zixiu; Whitt, Michael A.
CORPORATE SOURCE: Departments of Neurosurgery, Pathology and Laboratory Medicine, and Molecular Sciences, The University of Tennessee Health Science Center, Memphis, TN, USA
SOURCE: Journal of Neurosurgery (2004), 100(6), 1049-1059
CODEN: JONSAC; ISSN: 0022-3085
PUBLISHER: American Association of Neurological Surgeons
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Recombinant vesicular stomatitis virus vectors as oncolytic agents in the treatment of high-grade gliomas in an organotypic brain tissue slice-glioma coculture model
REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 17 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:363266 CAPLUS
DOCUMENT NUMBER: 140:417458

TITLE: Syncytia Induction Enhances the Oncolytic Potential of Vesicular Stomatitis Virus in Virotherapy for Cancer
AUTHOR(S): Ebert, Oliver; Shinozaki, Katsunori; Kournioti, Chryssanthi; Park, Man-Seong; Garcia-Sastre, Adolfo; Woo, Savio L. C.
CORPORATE SOURCE: Carl C. Icahn Center for Gene Therapy and Molecular Medicine, Mount Sinai School of Medicine, New York, NY, 10029, USA
SOURCE: Cancer Research (2004), 64(9), 3265-3270
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Syncytia Induction Enhances the Oncolytic Potential of Vesicular Stomatitis Virus in Virotherapy for Cancer
REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 18 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:190744 CAPLUS
DOCUMENT NUMBER: 140:350159
TITLE: Oncolysis of Multifocal Hepatocellular Carcinoma in the Rat Liver by Hepatic Artery Infusion of Vesicular Stomatitis Virus
AUTHOR(S): Shinozaki, Katsunori; Ebert, Oliver; Kournioti, Chryssanthi; Tai, Yun-Sheng; Woo, Savio L. C.
CORPORATE SOURCE: Carl C. Icahn Center for Gene Therapy and Molecular Medicine, Mount Sinai School of Medicine, New York, NY, 10029-6574, USA
SOURCE: Molecular Therapy (2004), 9(3), 368-376
CODEN: MTOHCK; ISSN: 1525-0016
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Oncolysis of Multifocal Hepatocellular Carcinoma in the Rat Liver by Hepatic Artery Infusion of Vesicular Stomatitis Virus
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 19 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:115829 CAPLUS
DOCUMENT NUMBER: 140:179252
TITLE: Defective translational control facilitates vesicular

stomatitis virus oncolysis
AUTHOR(S): Balachandran, Siddharth; Barber, Glen N.
CORPORATE SOURCE: Department of Microbiology and Immunology and Sylvester Comprehensive Cancer Center, University of Miami School of Medicine, Miami, FL, 33136, USA
SOURCE: Cancer Cell (2004), 5(1), 51-65
CODEN: CCAECI; ISSN: 1535-6108
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Defective translational control facilitates vesicular stomatitis virus oncolysis
REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 20 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:982369 CAPLUS
DOCUMENT NUMBER: 140:58249
TITLE: The Oncolytic Effect of Recombinant Vesicular Stomatitis Virus Is Enhanced by Expression of the Fusion Cytosine Deaminase/Uracil Phosphoribosyltransferase Suicide Gene
AUTHOR(S): Porosnicu, Mercedes; Mian, Abdul; Barber, Glen N.
CORPORATE SOURCE: Department of Microbiology and Immunology and Sylvester Comprehensive Cancer Center, University of Miami School of Medicine, Miami, FL, USA
SOURCE: Cancer Research (2003), 63(23), 8366-8376
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English
TI The Oncolytic Effect of Recombinant Vesicular Stomatitis Virus Is Enhanced by Expression of the Fusion Cytosine Deaminase/Uracil Phosphoribosyltransferase Suicide Gene
REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 21 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:940651 CAPLUS
DOCUMENT NUMBER: 140:58350
TITLE: Vesicular stomatitis virus expressing a chimeric Sindbis glycoprotein containing an Fc antibody binding domain targets to Her2/neu overexpressing breast cancer cells

AUTHOR(S): Bergman, Ira; Whitaker-Dowling, Patricia; Gao, Yanhua; Griffin, Judith A.; Watkins, Simon C.
CORPORATE SOURCE: Departments of Pediatrics, Neuorlogy and Immunology, University of Pittsburgh School of Medicine, Pittsburgh, PA, 15213, USA
SOURCE: Virology (2003), 316(2), 337-347
CODEN: VIRLAX; ISSN: 0042-6822
PUBLISHER: Elsevier Science
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Vesicular stomatitis virus expressing a chimeric Sindbis glycoprotein containing an Fc antibody binding domain targets to Her2/neu overexpressing breast cancer cells
REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 22 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:873984 CAPLUS
DOCUMENT NUMBER: 140:122323
TITLE: VSV strains with defects in their ability to shutdown innate immunity are potent systemic anti-cancer agents
AUTHOR(S): Stojdl, David F.; Lichty, Brian D.; ten Oever, Benjamin R.; Paterson, Jennifer M.; Power, Anthony T.; Knowles, Shane; Marius, Ricardo; Reynard, Jennifer; Poliquin, Laurent; Atkins, Harold; Brown, Earl G.; Durbin, Russell K.; Durbin, Joan E.; Hiscott, John; Bell, John C.
CORPORATE SOURCE: Ottawa Regional Cancer Centre Research Laboratories, Ottawa, ON, K1H 8L6, Can.
SOURCE: Cancer Cell (2003), 4(4), 263-275
CODEN: CCAECI; ISSN: 1535-6108
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal
LANGUAGE: English
TI VSV strains with defects in their ability to shutdown innate immunity are potent systemic anti-cancer agents
REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 23 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:873978 CAPLUS
DOCUMENT NUMBER: 140:121916
TITLE: Vesicular stomatitis virus: An exciting new

therapeutic oncolytic virus candidate for cancer or just another chapter from Field's virology?
AUTHOR(S): Giedlin, Martin A.; Cook, David N.; Dubensky, Thomas W., Jr.
CORPORATE SOURCE: Cancer Research, Cerus Corporation, Concord, CA, 94520, USA
SOURCE: Cancer Cell (2003), 4(4), 241-243
CODEN: CCAECI; ISSN: 1535-6108
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
TI Vesicular stomatitis virus: An exciting new therapeutic oncolytic virus candidate for cancer or just another chapter from Field's virology?
REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 24 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:668101 CAPLUS
DOCUMENT NUMBER: 140:104590
TITLE: Oncolysis of hepatic metastasis of colorectal cancer by recombinant vesicular stomatitis virus in immune-competent mice
AUTHOR(S): Huang, Tian-Gui; Ebert, Oliver; Shinozaki, Katsunori; Garcia-Sastre, Adolfo; Woo, Savio L. C.
CORPORATE SOURCE: Carl C. Icahn Center for Gene Therapy and Molecular Medicine, Mount Sinai School of Medicine, New York, NY, 10029-6574, USA
SOURCE: Molecular Therapy (2003), 8(3), 434-440
CODEN: MTOHCK; ISSN: 1525-0016
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Oncolysis of hepatic metastasis of colorectal cancer by recombinant vesicular stomatitis virus in immune-competent mice
REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 25 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:620161 CAPLUS
DOCUMENT NUMBER: 139:244498
TITLE: Development of recombinant vesicular stomatitis viruses that exploit defects in host defense to augment specific oncolytic activity
AUTHOR(S): Obuchi, Masatsugu; Fernandez, Marilyn; Barber, Glen N.

CORPORATE SOURCE: Department of Microbiology and Immunology and Sylvester Comprehensive Cancer Center, University of Miami School of Medicine, Miami, FL, 33136, USA
SOURCE: Journal of Virology (2003), 77(16), 8843-8856
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Development of recombinant vesicular stomatitis viruses that exploit defects in host defense to augment specific oncolytic activity
REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 26 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:505884 CAPLUS
DOCUMENT NUMBER: 139:190850
TITLE: Oncolytic vesicular stomatitis virus for treatment of orthotopic hepatocellular carcinoma in immune-competent rats
AUTHOR(S): Ebert, Oliver; Shinozaki, Katsunori; Huang, Tian-Gui; Savontaus, Mikko J.; Garcia-Sastre, Adolfo; Woo, Savio L. C.
CORPORATE SOURCE: Carl C. Icahn Institute for Gene Therapy and Molecular Medicine, Mount Sinai School of Medicine, New York, NY, 10029-6574, USA
SOURCE: Cancer Research (2003), 63(13), 3605-3611
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Oncolytic vesicular stomatitis virus for treatment of orthotopic hepatocellular carcinoma in immune-competent rats
REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 27 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:440343 CAPLUS
DOCUMENT NUMBER: 139:227186
TITLE: Vesicular stomatitis virus selectively inducing cytolysis of hepatocellular carcinoma cell line
AUTHOR(S): Tan, Longyi; Zhang, Li; Chen, Jie; Hu, Haiqing; Lin, Shuying; Wang, Hao
CORPORATE SOURCE: Department of Laboratory Diagnosis, Changzheng Hospital, Second Military Medical University,

Shanghai, 200003, Peop. Rep. China
SOURCE: Dier Junyi Daxue Xuebao (2003), 24(1), 67-69
CODEN: DJXUE5; ISSN: 0258-879X
PUBLISHER: Dier Junyi Daxue Xuebao Bianjibu
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
TI Vesicular stomatitis virus selectively inducing cytolysis of hepatocellular carcinoma cell line

L3 ANSWER 28 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:57860 CAPLUS
DOCUMENT NUMBER: 138:117637
TITLE: Recombinant vesicular stomatitis virus (VSV) vector for the treatment of tumor cells
INVENTOR(S): Barber, Glen
PATENT ASSIGNEE(S): University of Miami, USA
SOURCE: PCT Int. Appl., 85 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003005964	A2	20030123	WO 2002-US22146	20020711
WO 2003005964	A3	20030424		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2452517	AA	20030123	CA 2002-2452517	20020711
US 2003044386	A1	20030306	US 2002-194594	20020711
EP 1411880	A2	20040428	EP 2002-749985	20020711
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
JP 2004537305	T2	20041216	JP 2003-511773	20020711

PRIORITY APPLN. INFO.: US 2001-304125P P 20010711
WO 2002-US22146 W 20020711
TI Recombinant vesicular stomatitis virus (VSV) vector for the

treatment of tumor cells

L3 ANSWER 29 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:12214 CAPLUS
DOCUMENT NUMBER: 136:288667
TITLE: Genetically engineered vesicular stomatitis virus in gene therapy: application for treatment of malignant disease
AUTHOR(S): Fernandez, Marilyn; Porosnicu, Mercedes; Markovic, Dubravka; Barber, Glen N.
CORPORATE SOURCE: Department of Microbiology and Immunology and Sylvester Comprehensive Cancer Center, University of Miami School of Medicine, Miami, FL, 33136, USA
SOURCE: Journal of Virology (2002), 76(2), 895-904
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Genetically engineered vesicular stomatitis virus in gene therapy: application for treatment of malignant disease
REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 30 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:205204 CAPLUS
DOCUMENT NUMBER: 135:2622
TITLE: Oncolytic activity of vesicular stomatitis virus is effective against tumors exhibiting aberrant p53, Ras, or Myc function and involves the induction of apoptosis
AUTHOR(S): Balachandran, Siddharth; Porosnicu, Mercedes; Barber, Glen N.
CORPORATE SOURCE: Department of Microbiology and Immunology and Sylvester Comprehensive Cancer Center, University of Miami School of Medicine, Miami, FL, 33136, USA
SOURCE: Journal of Virology (2001), 75(7), 3474-3479
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Oncolytic activity of vesicular stomatitis virus is effective against tumors exhibiting aberrant p53, Ras, or Myc function and involves the induction of apoptosis
REFERENCE COUNT: 19 THERE ARE 19 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 31 OF 58 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:504637 CAPLUS
DOCUMENT NUMBER: 133:191813
TITLE: Exploiting tumor-specific defects in the interferon pathway with a previously unknown oncolytic virus
AUTHOR(S): Stojdl, David F.; Lichty, Brian; Knowles, Shane; Marius, Ricardo; Atkins, Harold; Sonenberg, Nahum; Bell, John C.
CORPORATE SOURCE: Ottawa Regional Cancer Centre Research Laboratories, Ottawa, ON, K1H 8L6, Can.
SOURCE: Nature Medicine (New York) (2000), 6(7), 821-825
CODEN: NAMEFI; ISSN: 1078-8956
PUBLISHER: Nature America Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
TI Exploiting tumor-specific defects in the interferon pathway with a previously unknown oncolytic virus
REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 32 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2006:479982 BIOSIS
DOCUMENT NUMBER: PREV200600471262
TITLE: Gene therapy strategies for hepatocellular carcinoma.
AUTHOR(S): Hwang, Lih-Hwa [Reprint Author]
CORPORATE SOURCE: Natl Taiwan Univ Hosp, Hepatitis Res Ctr, 7,Chungshan S Rd, Taipei 10016, Taiwan
lihhwa@ha.mc.ntu.edu.tw
SOURCE: Journal of Biomedical Science, (JUL 2006) Vol. 13, No. 4, pp. 453-468.
ISSN: 1021-7770.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Sep 2006
Last Updated on STN: 20 Sep 2006
TI Gene therapy strategies for hepatocellular carcinoma.

L3 ANSWER 33 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on

STN
ACCESSION NUMBER: 2006:443605 BIOSIS
DOCUMENT NUMBER: PREV200600441299
TITLE: Vesicular stomatitis virus induces apoptosis in the Wong-Kilbourne derivative of the Chang conjunctival cell line.
AUTHOR(S): Gallyas, Eva; Seprenyi, Gyorgy; Sonkoly, Eniko; Mandi, Yvette; Kemeny, Lajos; Megyeri, Klara [Reprint Author]
CORPORATE SOURCE: Univ Szeged, Dept Med Microbiol and Immunolbiol, Dom Ter 10, H-6720 Szeged, Hungary
megyeri@comser.szote.u-szeged.hu
SOURCE: Graefe's Archive for Clinical and Experimental Ophthalmology, (JUN 2006) Vol. 244, No. 6, pp. 717-724.
CODEN: GACODL. ISSN: 0721-832X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Sep 2006
Last Updated on STN: 6 Sep 2006
TI Vesicular stomatitis virus induces apoptosis in the Wong-Kilbourne derivative of the Chang conjunctival cell line.

L3 ANSWER 34 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2006:215422 BIOSIS
DOCUMENT NUMBER: PREV200600215554
TITLE: Oncolytic activity of vesicular stomatitis virus in primary adult T-cell leukemia.
AUTHOR(S): Cesaire, R.; Oliere, S.; Sharif-Askari, E.; Loignon, M.; Lezin, A.; Olindo, S.; Panelatti, G.; Kazanji, M.; Aloyz, R.; Panasci, L.; Bell, J. C.; Hiscott, J. [Reprint Author]
CORPORATE SOURCE: McGill Univ, Jewish Gen Hosp, Lady Davis Inst Med Res, Mol Oncol Grp, Room 526,3755 Cote Ste Catherine Rd, Montreal, PQ H3T 1E2, Canada
john.hiscott@mcgill.ca
SOURCE: Oncogene, (JAN 2006) Vol. 25, No. 3, pp. 349-358.
CODEN: ONCNES. ISSN: 0950-9232.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 29 Mar 2006
Last Updated on STN: 29 Mar 2006
TI Oncolytic activity of vesicular stomatitis virus in primary adult T-cell leukemia.

L3 ANSWER 35 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2006:95255 BIOSIS
DOCUMENT NUMBER: PREV200600098446
TITLE: VSV-tumor selective replication and protein translation.
AUTHOR(S): Barber, Glen N. [Reprint Author]
CORPORATE SOURCE: Univ Miami, Sch Med, Dept Microbiol and Immunol, Sylvester Comprehens Canc Ctr, Room 511,Papanicolaou Bldg,1550 NW 10th St M710, Miami, FL 33136 USA
gbarber@med.miami.edu
SOURCE: Oncogene, (NOV 21 2005) Vol. 24, No. 52, pp. 7710-7719.
CODEN: ONCNES. ISSN: 0950-9232.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Feb 2006
Last Updated on STN: 1 Feb 2006
TI VSV-tumor selective replication and protein translation.

L3 ANSWER 36 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2006:39669 BIOSIS
DOCUMENT NUMBER: PREV200600039249
TITLE: Prophylactic alpha interferon treatment increases the therapeutic index of oncolytic vesicular stomatitis virus virotherapy for advanced hepatocellular carcinoma in immune-competent rats.
AUTHOR(S): Shinozaki, Katsunori; Ebert, Oliver; Suriawinata, Arief; Thung, Swan N.; Woo, Savio L. C. [Reprint Author]
CORPORATE SOURCE: Mt Sinai Sch Med, Dept Gene and Cell Med, 1 Gustave L Levy Pl,Box 1496, New York, NY 10029 USA
savio.woo@mssm.edu
SOURCE: Journal of Virology, (NOV 2005) Vol. 79, No. 21, pp. 13705-13713.
CODEN: JOVIAM. ISSN: 0022-538X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 28 Dec 2005
Last Updated on STN: 28 Dec 2005
TI Prophylactic alpha interferon treatment increases the therapeutic index of oncolytic vesicular stomatitis virus virotherapy for advanced

hepatocellular carcinoma in immune-competent rats.

L3 ANSWER 37 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2005:290643 BIOSIS
DOCUMENT NUMBER: PREV200510081946
TITLE: Targeting human glioblastoma cells: Comparison of nine viruses with oncolytic potential.
AUTHOR(S): Wollmann, Guido; Tattersall, Peter; van den Pol, Anthony N. [Reprint Author]
CORPORATE SOURCE: Yale Univ, Sch Med, Dept Neurosurg, 333 Cedar St, New Haven, CT 06520 USA
anthony.vandenpol@yale.edu
SOURCE: Journal of Virology, (MAY 2005) Vol. 79, No. 10, pp. 6005-6022.
CODEN: JOVIAM. ISSN: 0022-538X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Aug 2005
Last Updated on STN: 4 Aug 2005
TI Targeting human glioblastoma cells: Comparison of nine viruses with oncolytic potential.

L3 ANSWER 38 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2005:223834 BIOSIS
DOCUMENT NUMBER: PREV200510011423
TITLE: Systemic therapy of experimental breast cancer metastases by mutant vesicular stomatitis virus in immune-competent mice.
AUTHOR(S): Ebert, Oliver; Harbaran, Sonal; Shinozaki, Katsunori; Woo, Savio L. C. [Reprint Author]
CORPORATE SOURCE: Mt Sinai Sch Med, Dept Gene and Cell Med, 1 Gustave L Levy
Pl,Box 1496, New York, NY 10029 USA
savio.woo@mssm.edu
SOURCE: Cancer Gene Therapy, (APR 2005) Vol. 12, No. 4, pp. 350-358.
ISSN: 0929-1903.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jun 2005
Last Updated on STN: 16 Jun 2005
TI Systemic therapy of experimental breast cancer metastases by mutant

vesicular stomatitis virus in immune-competent mice.

L3 ANSWER 39 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2005:215738 BIOSIS
DOCUMENT NUMBER: PREV200510004369
TITLE: Treatment of multi-focal colorectal carcinoma metastatic to the liver of immune-competent and syngeneic rats by hepatic artery infusion of oncolytic vesicular stomatitis virus.
AUTHOR(S): Shinozaki, Katsunori; Ebert, Oliver; Woo, Savio L. C. [Reprint Author]
CORPORATE SOURCE: CUNY Mt Sinai Sch Med, Dept Gene and Cell Med, 1 Gustave L Levy Pl,Box 1496, New York, NY 10029 USA
savio.woo@mssm.edu
SOURCE: International Journal of Cancer, (APR 20 2005) Vol. 114, No. 4, pp. 659-664.
CODEN: IJCNAW. ISSN: 0020-7136.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Jun 2005
Last Updated on STN: 10 Jun 2005
TI Treatment of multi-focal colorectal carcinoma metastatic to the liver of immune-competent and syngeneic rats by hepatic artery infusion of oncolytic vesicular stomatitis virus.

L3 ANSWER 40 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2005:136795 BIOSIS
DOCUMENT NUMBER: PREV200500135625
TITLE: Vesicular stomatitis virus as an oncolytic vector.
AUTHOR(S): Barber, Glen N. [Reprint Author]
CORPORATE SOURCE: Sch MedSylvester Comprehens Canc CtrDept Microbiol and Immunol, Miami Univ, Rm 511 Papanicolaou Bldg,1550 NW 10th St M710, Miami, FL, 33136, USA
gbarber@med.miami.edu
SOURCE: Viral Immunology, (Winter 2004) Vol. 17, No. 4, pp. 516-527. print.
CODEN: VIIMET. ISSN: 0882-8245.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English

ENTRY DATE: Entered STN: 6 Apr 2005
Last Updated on STN: 6 Apr 2005
TI Vesicular stomatitis virus as an oncolytic vector.

L3 ANSWER 41 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2005:102904 BIOSIS
DOCUMENT NUMBER: PREV200500092966
TITLE: Eradication of advanced hepatocellular carcinoma in rats via repeated hepatic arterial infusions of recombinant VSV.
AUTHOR(S): Shinozaki, Katsunori; Ebert, Ohver; Woo, Savio L. C. [Reprint Author]
CORPORATE SOURCE: Dept Gene and Cell Med, CUNY Mt Sinai Sch Med, 1 Gustave L Levy Pl,Box 1496, New York, NY, 10029, USA
savio.woo@mssm.edu
SOURCE: Hepatology, (January 2005) Vol. 41, No. 1, pp. 196-203. print.
ISSN: 0270-9139 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Mar 2005
Last Updated on STN: 9 Mar 2005
TI Eradication of advanced hepatocellular carcinoma in rats via repeated hepatic arterial infusions of recombinant VSV.

L3 ANSWER 42 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2005:82408 BIOSIS
DOCUMENT NUMBER: PREV200500076460
TITLE: Sensitivity of prostate tumors to wild type and M protein mutant vesicular stomatitis viruses.
AUTHOR(S): Ahmed, Maryam [Reprint Author]; Cramer, Scott D.; Lyles, Douglas S.
CORPORATE SOURCE: Sch MedDept Biochem, Wake Forest Univ, Med Ctr Blvd, Winston Salem, NC, 27157, USA
mahmed@wfubmc.edu
SOURCE: Virology, (December 5 2004) Vol. 330, No. 1, pp. 34-49. print.
ISSN: 0042-6822 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Feb 2005

Last Updated on STN: 23 Feb 2005
TI Sensitivity of prostate tumors to wild type and M protein mutant vesicular stomatitis viruses.

L3 ANSWER 43 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:463465 BIOSIS
DOCUMENT NUMBER: PREV200400464964
TITLE: Induction of apoptosis and tumor regression by vesicular stomatitis virus in the presence of gemcitabine in lung cancer.
AUTHOR(S): Li, Qiu; Wei, Yu-quan [Reprint Author]; Wen, Yan-jun; Zhao, Xia; Tian, Ling; Yang, Li; Mao, Yong-qiu; Kan, Bing; Wu, Yang; Ding, Zhen-yu; Deng, Hong-Xin; Li, Jiong; Luo, Yan; Li, Hong-; He, Qiu-ming; Su, Jing-mei; Xiao, Fei; Zou, Chun-Hua; Fu, Chun-Hua; Xie, Xing-jiang; Yi, Tao; Tan, Guang-Hong; Wang, Lian; Chen, Jing; Liu, Jian; Gao, Zhen-Nan
CORPORATE SOURCE: W China Med SchW China HospLab Biotherapy Human Dis,Minist Educ, Sichuan Univ, Guo Xue Xiang 37, Chengdu, Sichuan, 610041, China
yuquanwei@vip.sina.com
SOURCE: International Journal of Cancer, (October 20 2004) Vol. 112, No. 1, pp. 143-149. print.
CODEN: IJCNAW. ISSN: 0020-7136.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Dec 2004
Last Updated on STN: 1 Dec 2004
TI Induction of apoptosis and tumor regression by vesicular stomatitis virus in the presence of gemcitabine in lung cancer.

L3 ANSWER 44 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:450979 BIOSIS
DOCUMENT NUMBER: PREV200400452923
TITLE: Replication and cytopathic effect of oncolytic vesicular stomatitis virus in hypoxic tumor cells in vitro and in vivo.
AUTHOR(S): Connor, John H. [Reprint Author]; Naczki, Christine; Koumenis, Costas; Lyles, Douglas S.
CORPORATE SOURCE: Sch MedDept Microbiol and Immunol, Wake Forest Univ, Med

Ctr Blvd, Winston Salem, NC, 27157, USA
jconnor@wfubmc.edu
SOURCE: Journal of Virology, (September 2004) Vol. 78, No. 17, pp. 8960-8970. print.
ISSN: 0022-538X (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Nov 2004
Last Updated on STN: 24 Nov 2004
TI Replication and cytopathic effect of oncolytic vesicular stomatitis virus in hypoxic tumor cells in vitro and in vivo.

L3 ANSWER 45 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:423955 BIOSIS
DOCUMENT NUMBER: PREV200400419862
TITLE: Vesicular stomatitis virus: A potential therapeutic virus for the treatment of hematologic malignancy.
AUTHOR(S): Lichty, Brian D.; Stojdl, David F.; Taylor, Rebecca A.; Miller, Leigh; Frenkel, Irina; Atkins, Harold; Bell, John C. [Reprint Author]
CORPORATE SOURCE: Res Labs, Ottawa Reg Canc Ctr, 503 Smyth Rd, Ottawa, ON, K1H 1C4, Canada
jbell@ohri.ca
SOURCE: Human Gene Therapy, (September 2004) Vol. 15, No. 9, pp. 821-831. print.
ISSN: 1043-0342 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 3 Nov 2004
Last Updated on STN: 3 Nov 2004
TI Vesicular stomatitis virus: A potential therapeutic virus for the treatment of hematologic malignancy.

L3 ANSWER 46 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:293987 BIOSIS
DOCUMENT NUMBER: PREV200400292339
TITLE: Syncytia induction enhances the oncolytic potential of vesicular stomatitis virus in virotherapy for cancer.
AUTHOR(S): Ebert, Oliver; Shinozaki, Katsunori; Kournioti, Chryssanthi; Park, Man-Seong; Garcia-Sastre, Adolfo; Woo, Savio L. C. [Reprint Author]

CORPORATE SOURCE: Carl C Icahn Ctr Gene Therapy and Mol Med, Mt Sinai Sch Med, 1 Gustave L Levy Pl,Box 1496, New York, NY, 10029, USA savio.woo@mssm.edu
SOURCE: Cancer Research, (May 1 2004) Vol. 64, No. 9, pp. 3265-3270. print.
ISSN: 0008-5472 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Jun 2004
Last Updated on STN: 23 Jun 2004
TI Syncytia induction enhances the oncolytic potential of vesicular stomatitis virus in virotherapy for cancer.

L3 ANSWER 47 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:204281 BIOSIS
DOCUMENT NUMBER: PREV200400204824
TITLE: Using a novel organotypic brain slice - glioma CO - culture model in studying recombinant VSV vectors as oncolytic agents.
AUTHOR(S): Zhou, Q. [Reprint Author]; Duntsch, C. D. [Reprint Author]; Jayakar, H. R.; Weimar, J. D. [Reprint Author]; Whitt, M. A.
CORPORATE SOURCE: Dept. of Neurosurgery, The Univ. Tennessee Hlth. Sci. Ctr., Memphis, TN, USA
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 752.15. http://sfn.scholarone.com. e-file.
Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004
TI Using a novel organotypic brain slice - glioma CO - culture model in studying recombinant VSV vectors as oncolytic agents.

L3 ANSWER 48 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:47308 BIOSIS
DOCUMENT NUMBER: PREV200400039977
TITLE: The oncolytic effect of recombinant vesicular

stomatitis virus is enhanced by expression of the fusion cytosine deaminase/uracil phosphoribosyltransferase suicide gene.
AUTHOR(S): Porosnicu, Mercedes; Mian, Abdul; Barber, Glen N. [Reprint Author]
CORPORATE SOURCE: University of Miami School of Medicine, 1550 North West 10th Avenue, Room 514, Papanicolaou Building, M710, Miami, FL, 33136, USA
gbarber@med.miami.edu
SOURCE: Cancer Research, (December 1 2003) Vol. 63, No. 23, pp. 8366-8376. print.
ISSN: 0008-5472 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Jan 2004
Last Updated on STN: 14 Jan 2004
TI The oncolytic effect of recombinant vesicular stomatitis virus is enhanced by expression of the fusion cytosine deaminase/uracil phosphoribosyltransferase suicide gene.

L3 ANSWER 49 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:30871 BIOSIS
DOCUMENT NUMBER: PREV200400023482
TITLE: Vesicular stomatitis virus expressing a chimeric Sindbis glycoprotein containing an Fc antibody binding domain targets to Her2/neu overexpressing breast cancer cells.
AUTHOR(S): Bergman, Ira [Reprint Author]; Whitaker-Dowling, Patricia; Gao, Yanhua; Griffin, Judith A.; Watkins, Simon C.
CORPORATE SOURCE: Children's Hospital of Pittsburgh, 3705 Fifth Avenue, Pittsburgh, PA, 15213, USA
ira.bergman@chp.edu
SOURCE: Virology, (November 25 2003) Vol. 316, No. 2, pp. 337-347. print.
ISSN: 0042-6822 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 31 Dec 2003
Last Updated on STN: 31 Dec 2003
TI Vesicular stomatitis virus expressing a chimeric Sindbis glycoprotein containing an Fc antibody binding domain targets to Her2/neu overexpressing breast cancer cells.

L3 ANSWER 50 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on

STN
ACCESSION NUMBER: 2004:3866 BIOSIS
DOCUMENT NUMBER: PREV200400006931
TITLE: VSV strains with defects in their ability to shutdown innate immunity are potent systemic anti-cancer agents.
AUTHOR(S): Stojdl, David F.; Lichty, Brian D.; tenOever, Benjamin R.; Paterson, Jennifer M.; Power, Anthony T.; Knowles, Shane; Marius, Ricardo; Reynard, Jennifer; Poliquin, Laurent; Atkins, Harold; Brown, Earl G.; Durbin, Russell K.; Durbin, Joan E.; Hiscott, John; Bell, John C. [Reprint Author]
CORPORATE SOURCE: Research Laboratories, Ottawa Regional Cancer Centre, 501 Smyth Road, Ottawa, ON, K1H 8L6, Canada
John.bell@orcc.on.ca
SOURCE: Cancer Cell, (October 2003) Vol. 4, No. 4, pp. 263-275. print.
ISSN: 1535-6108 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Dec 2003
Last Updated on STN: 17 Dec 2003
TI VSV strains with defects in their ability to shutdown innate immunity are potent systemic anti-cancer agents.

L3 ANSWER 51 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2004:3855 BIOSIS
DOCUMENT NUMBER: PREV200400006925
TITLE: Vesicular stomatitis virus: An exciting new therapeutic oncolytic virus candidate for cancer or just another chapter from Field's Virology?.
AUTHOR(S): Giedlin, Martin A. [Reprint Author]; Cook, David N. [Reprint Author]; Dubensky, Thomas W. [Reprint Author]
CORPORATE SOURCE: Cancer Research, Cerus Corporation, 2411 Stanwell Drive, Concord, CA, 94520, USA
tom_dubensky@ceruscorp.com
SOURCE: Cancer Cell, (October 2003) Vol. 4, No. 4, pp. 241-243. print.
ISSN: 1535-6108 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Dec 2003
Last Updated on STN: 17 Dec 2003
TI Vesicular stomatitis virus: An exciting new therapeutic oncolytic virus candidate for cancer or just another chapter from Field's Virology?.

L3 ANSWER 52 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2003:432731 BIOSIS
DOCUMENT NUMBER: PREV200300432731
TITLE: Development of recombinant vesicular stomatitis viruses that exploit defects in host defense to augment specific oncolytic activity.
AUTHOR(S): Obuchi, Masatsugu; Fernandez, Marilyn; Barber, Glen N. [Reprint Author]
CORPORATE SOURCE: University of Miami School of Medicine, 1550 NW 10th Ave., Rm. 511, Papanicolaou Building, M710, Miami, FL, 33136, USA gbarber@med.miami.edu
SOURCE: Journal of Virology, (August 2003) Vol. 77, No. 16, pp. 8843-8856. print.
ISSN: 0022-538X (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Sep 2003
Last Updated on STN: 17 Sep 2003
TI Development of recombinant vesicular stomatitis viruses that exploit defects in host defense to augment specific oncolytic activity.

L3 ANSWER 53 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2003:346206 BIOSIS
DOCUMENT NUMBER: PREV200300346206
TITLE: Oncolytic vesicular stomatitis virus for treatment of orthotopic hepatocellular carcinoma in immune-competent rats.
AUTHOR(S): Ebert, Oliver; Shinozaki, Katsunori; Huang, Tian-Gui; Savontaus, Mikko J.; Garcia-Sastre, Adolfo; Woo, Savio L. C. [Reprint Author]
CORPORATE SOURCE: Carl C. Icahn Institute for Gene Therapy and Molecular Medicine, Mount Sinai School of Medicine, One Gustave L. Levy Place, Box 1496, New York, NY, 10029-6574, USA savio.woo@mssm.edu
SOURCE: Cancer Research, (July 1 2003) Vol. 63, No. 13, pp. 3605-3611. print.
ISSN: 0008-5472 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 30 Jul 2003

Last Updated on STN: 30 Jul 2003
TI Oncolytic vesicular stomatitis virus for treatment of orthotopic hepatocellular carcinoma in immune-competent rats.

L3 ANSWER 54 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:215362 BIOSIS
DOCUMENT NUMBER: PREV200200215362
TITLE: Cytolytic viruses as potential anti-cancer agents.
AUTHOR(S): Ring, Christopher J. A. [Reprint author]
CORPORATE SOURCE: Gene Interference, Medicines Research Centre, Glaxo SmithKline Research and Development, Gunnels Wood Road, Stevenage, Herts, SG1 2NY, UK
cjr48991@gsk.com
SOURCE: Journal of General Virology, (March, 2002) Vol. 83, No. 3, pp. 491-502. print.
CODEN: JGVIAY. ISSN: 0022-1317.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Mar 2002
Last Updated on STN: 27 Mar 2002
TI Cytolytic viruses as potential anti-cancer agents.

L3 ANSWER 55 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:143666 BIOSIS
DOCUMENT NUMBER: PREV200200143666
TITLE: Genetically engineered vesicular stomatitis virus in gene therapy: Application for treatment of malignant disease.
AUTHOR(S): Fernandez, Marilyn; Porosnicu, Mercedes; Markovic, Dubravka; Barber, Glen N. [Reprint author]
CORPORATE SOURCE: University of Miami School of Medicine, 1550 NW 10th Ave., Rm. 511, Papanicolaou Building, M710, Miami, FL, 33136, USA
gbarber@med.miami.edu
SOURCE: Journal of Virology, (January, 2002) Vol. 76, No. 2, pp. 895-904. print.
CODEN: JOVIAM. ISSN: 0022-538X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Feb 2002
Last Updated on STN: 26 Feb 2002
TI Genetically engineered vesicular stomatitis virus in gene therapy:

Application for treatment of malignant disease.

L3 ANSWER 56 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2001:242607 BIOSIS
DOCUMENT NUMBER: PREV200100242607
TITLE: Oncolytic activity of vesicular stomatitis virus is effective against tumors exhibiting aberrant p53, ras, or myc function and involves the induction of apoptosis.
AUTHOR(S): Balachandran, Siddharth; Porosnicu, Mercedes; Barber, Glen N. [Reprint author]
CORPORATE SOURCE: University of Miami School of Medicine, 1550 NW 10th Ave., Rm. 514, Papanicolaou Building, M710, Miami, FL, 33136, USA gbarber@med.miami.edu
SOURCE: Journal of Virology, (April, 2001) Vol. 75, No. 7, pp. 3474-3479. print.
CODEN: JOVIAM. ISSN: 0022-538X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 16 May 2001
Last Updated on STN: 19 Feb 2002
TI Oncolytic activity of vesicular stomatitis virus is effective against tumors exhibiting aberrant p53, ras, or myc function and involves the induction of apoptosis.

L3 ANSWER 57 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2001:53684 BIOSIS
DOCUMENT NUMBER: PREV200100053684
TITLE: Vesicular stomatitis virus (VSV) therapy of tumors.
AUTHOR(S): Balachandran, Siddharth; Barber, Glen N. [Reprint author]
CORPORATE SOURCE: University of Miami School of Medicine, 1550 NW 10th Ave., 514 Papanicolaou Bldg., Miami, FL, 33136, USA gbarber@med.miami.edu
SOURCE: IUBMB Life, (August, 2000) Vol. 50, No. 2, pp. 135-138. print.
ISSN: 1521-6543.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Jan 2001
Last Updated on STN: 12 Feb 2002

TI Vesicular stomatitis virus (VSV) therapy of tumors.

L3 ANSWER 58 OF 58 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on
STN
ACCESSION NUMBER: 2000:428788 BIOSIS
DOCUMENT NUMBER: PREV200000428788
TITLE: Exploiting tumor-specific defects in the interferon pathway with a previously unknown oncolytic virus.
AUTHOR(S): Stojdl, David F.; Lichty, Brian; Knowles, Shane; Marius, Ricardo; Atkins, Harold; Sonenberg, Nahum; Bell, John C. [Reprint author]
CORPORATE SOURCE: Ottawa Regional Cancer Centre Research Laboratories, 501, Smyth Road, Ottawa, ON, K1H 8L6, Canada
SOURCE: Nature Medicine, (July, 2000) Vol. 6, No. 7, pp. 821-825. print.
ISSN: 1078-8956.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Oct 2000
Last Updated on STN: 10 Jan 2002
TI Exploiting tumor-specific defects in the interferon pathway with a previously unknown oncolytic virus.